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INCORPORATING THE INDEX OF OTOLARYNGOLOGY.

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XXXIII.

A STUDY OF RADICAL MASTOIDS.*

BY LEON E. WHITE, M. D.,

BOSTON, MASS.

This study was commenced in February, 1924, and completed, as far as operating on new cases was concerned, in March, 1927. It records all the radical mastoids, fifty-four in number, in fifteen months of actual but not consecutive service at the Massachusetts Eye and Ear Infirmary.

This work was undertaken to determine the value of various tests before operation and the relative merits of different methods of performing the operation and epidermatizing the radical cavity. We wished to observe the effects of the operation on hearing, freedom from discharge and intracranial complications. By checkups we hoped to learn something of the ultimate value of the radical mastoid and in what way, if any, we might improve our methods of handling these cases not only while in the hospital but more especially after their discharge to the outpatient.

All cases with three exceptions have been followed from one to three years after the operation. The majority of the patients returned for a final inspection of their radical

*Read before the American Otological Society, Inc., at Washington, D. C., on April 30, 1928.

cavities and hearing tests. We were thus able to check up on our results a year or more after the operation. This followup work is of the utmost importance among hospital cases, but it was found to have been neglected, at least at the Massachusetts Eye and Ear Infirmary, as many patients have gone out with beautifully epidermatized cavities only to return in a year or two with an ear filled with broken down debris and granulations. In private practice with cooperative patients these radical cavities are easily kept dry and clean for the patient's lifetime. Why can we not, with better and more systematic cooperation of our Social Service Department, obtain almost equally satisfactory results with our clinic cases?

Each year at the commencement of my service I gave the senior house surgeon a scroll of paper for tabulating the various tests. On the top of this scroll, which consisted of seven pages of manuscript paper, end to end, was typewritten the following: Name, Record number, Age, Ear affected, Duration, Cause, Previous operations, Nasal examination, X-ray of mastoid, X-ray of accessory sinuses, Character of discharge, Constant or intermittent, Cholesteatome, Middle ear examination, Mastoid examination, Previous treatment, Barany, Fundus, Visual fields, Reflexes, Hearing, Operator, Operation, Skin graft, Flap, Sutures, After treatment, Time in hospital, When dry and epidermatized, Complications, Remarks. I was especially interested to find if routine fundi examinations, visual fields and Barany tests would detect latent brain abscesses or commencing meningitis. The following summary of these tests furnishes the data for this study:

Age.—These fifty-four cases varied in age from four to fifty-seven years; ten were under ten years.

Ear Affected.—In sixteen the right ear only was involved; in twenty-eight the left ear; and in ten, both ears.

The Duration of the Discharge.—This varied from one to forty-five years. In twelve it was two years or less.

The Cause.—In twenty-eight it was not known. In eight it was attributed to measles. In six to scarlatina, and in three to tuberculosis. In four it followed the grip, and in three, pneumonia. One case was secondary to osteomyelitis

of the accessory sinuses; two had traumatic rupture of the drum membrane, and one, nasal diphtheria.

Previous Operations.—Twenty-four had tonsils and adenoids removed; sixteen, one or more simple mastoids; two had previous radicals on the ear operated upon, while three had radicals on the other ear. The ossicles had been taken out in two cases, and seventeen had aural polyps removed. None of the cases gave a history of resection of the septum, removal of turbinates or opening of any of the sinuses, although it is possible that some such work had been done but not noted.

Nasal Examination.—This was negative in forty cases. Five had deviations of the septum; one ethmoiditis, and eight a mucopurulent discharge. Three showed hypertrophied turbinates; two, spurs; one, ulceration of the septum, and one, nasal polyps.

X-rays of the Mastoids.—Twenty-three were sclerosed. Eleven showed postoperative cavities; eight marked destruction; eight a large cavity with surrounding sclerosis; four, a large cavity suggesting cholesteatome, and one, a sequestrum.

X-rays of the Accessory Sinuses.—These were negative in twenty-seven cases. The antrum showed considerable thickening of its lining membrane in twelve, and it contained pus in two; the ethmoids showed clouding in two; in eleven no record was made.

The Discharge.—This was noted as purulent with foul odor in thirty; purulent without odor in five; serous in six; mucopurulent in six; scanty in two; thick with cholesteatome in one; scanty with cholesteatome in one; not noted in three. It was constant in forty-seven, and intermittent in four.

Cholesteatome.—This was found in twenty-seven; that is, just one-half of these cases.

Examination of Middle Ear.—In thirteen, large polyps filled the canal, and a view of the drum membrane was not obtained. In fourteen the drum membrane was destroyed and the middle ear filled with granulations. In seven the canal wall was so swollen that the drum could not be made out; nine had marginal perforations with granulations; three destruction of the drum membrane with cholesteatome; three perforations in Shrapnell's membrane; one a bulging drum membrane, sag-

ging of the canal wall and granulations, evidently an acute infection of a chronic ear, and three had radical cavities filled with granulations. One case, probably an emergency, had no record of the middle ear.

Examination of the Mastoid.—Externally it was negative in half the cases. In twelve there was tenderness and swelling about the postoperative scar over the mastoid, while six simply showed postoperative scars. In six there was slight mastoid tenderness; two had mastoid tenderness and swelling, and one a postaural fistula.

Previous Treatment.—This is a very important item in every case undergoing a radical mastoid operation. If one could have seen these cases frequently and followed them over long periods of time, a limited few might have escaped the radical operation. Some came from long distances, which made it impossible for them to receive skilled treatment, so that it seemed safer to perform a radical rather than try prolonged treatment. There were but a few cases, however, except emergencies, that were operated upon until they had received a reasonable amount of local treatment. When a case had been coming to the clinic some months without showing improvement, it was felt that time would be wasted by further treatment and operation was advised. Children with tonsils or adenoids had them removed while undergoing preliminary treatment and cases showing antrum disease also underwent a preliminary operation to clean it up. A third of these cases had one or more previous mastoid operations which had failed to effect a cure.

The following summarizes the previous treatment: Three had been treated in the outpatient department for four or more years and had a simple mastoid operation. Two had been treated for seven or more years and had an ossilectomy. Seven had been treated from one to four years and had a simple mastoid. Six had been treated for five or more years and had two simple mastoid operations. One had been under treatment for ten years and had three simple mastoids and still another case had been under treatment for a similar period during which four simple mastoid operations had been performed. Four had radical mastoids but the cavities filled with granulations and scar tissue so that the operation had to

be repeated. Thirteen were treated from one to seventeen years by the routine methods in vogue in our outpatient department. Four cases had aural polyps removed and routine treatment for several years. Ten cases received no previous treatment at the Infirmary but were referred to us after being treated elsewhere. In three cases no note was made as to previous treatment.

The Barany Test.—This showed a normally functioning static labyrinth in thirty-four cases. There was no response in ten, but as all appeared to have some hearing the entire labyrinth could not have been involved. Six cases had delayed reactions. A diagnosis of serous labyrinthitis was recorded in one case and a lesion of the cerebellopontine angle in another, but the future course of this case showed no such lesions. Hypofunction of the vertical canals was noted in two cases. Outside of showing whether or not there was a normally functioning static labyrinth the Barany tests were essentially negative. None of these cases subsequently developed brain abscesses as far as we know, so that little more could have been expected from these tests. A negative report, however, is valuable.

The Fundi.—These were negative in all but one case and here slight tortuosity of the retinal veins with blurring of the nasal side of the disc were noted. This was of no diagnostic significance as the future course of the case proved. As none of these cases developed brain abscess or meningitis while on my service, we would naturally not expect to find papilledema. I believe it is a good practice to make routine examinations of the fundi so that we may have a record of its normal appearance should later complications develop.

Visual Fields.—These were normal in all and I doubt if they are of much importance unless intracranial complications develop.

The Reflexes.—These were also essentially negative. I did not think it advisable to do preliminary lumbar punctures as a routine procedure. It was done in one case where we suspected intracranial involvement but was negative.

Hearing Tests.—These were made before operation and on discharging the patient from the hospital; also when they were checked up one to three years after operation. The en-

tire paper might be devoted to this phase of the subject, but I will try to briefly summarize our findings. One-fourth of the cases showed more improvement in hearing on leaving the hospital, while an equal number showed a falling off in the hearing. The remaining half showed no change. Hearing tests were made on twenty-eight one to three years after operation. In none was there any improvement after leaving the hospital, but usually some diminution. The comparison of the hearing in these twenty-eight cases between what it was before operation and what it was found to be one or more years after operation, is extremely instructive. In six there was no change. In five there was marked improvement. The hearing for whispered voice in these showed the following changes: before operation 1/25, after 11/25; before 1/25, after 17/25; before 4/25, after 16/25; before 1/25, after 5/25; before 1/25, after 18/25. In four cases there was slight diminution in hearing, that is, from 1 to 3 feet in the spoken voice. In the remaining thirteen there was the following marked loss of hearing: From 7/35 to 1/35; from 10/35 to 1/35; from 3/25 to 1/25; from 11/35 to 1/35; from 18/25 to 1/35; from 5/25 to 1/25; from 13/25 to 1/35; from 4/25 to 1/25; from 10/35 to 1/45 from 18/25 to 1/45; from 10/25 to 1/35; from 7/35 to 1/35; from 10/35 to 1/45.

The denominators of these fractions represent respectively a 25-foot whisper, a 35-foot articulate voice, and a 45-foot loud voice. The numerators represent the number of feet that the patient hears. Taken as a whole there was rather marked impairment in the hearing after the patient left the hospital, probably due to cicatricial tissue about the stapes.

The Operators.—These comprised the men associated with me on my service. Dr. Philip Hammond as Chief of Service for most of this period gave us valuable help and in the several operations that he performed, set a very high standard which all of us tried to attain. I passed on all the cases before operation and watched their progress thereafter. There were fifteen operators. Six of these were house surgeons who operated under the guidance of some member of the staff. Dr. Lothrop led the operators by having eleven radicals to his credit. I was second with ten. Dr. White, Junior, per-

formed six; Doctors Hammond and Drury five each. The other operators with from one to three to their credit were Doctors Poirier, Simmons, Meltzer, Finck, Quincy, Brauner, Winkler, Richards, Ernland and Cave. Each operator selected the method he preferred of performing the operation and skin grafting the cavity.

The Radical Operation.—An effort was made to eradicate all the affected cells, to take down the facial ridge as low as possible consistent with the safety of the facial nerve, to remove the overhang of the epitympanic space; to take out the malleus and incus if present and curette the tympanic end of the eustachian tube. The tympanic ring was removed and the mucosa of the middle ear if diseased. These operations were performed in the quiescent stage. When the mastoid was at all acute, as frequently happens in recurrent mastoiditis, a simple mastoid was first performed. This was followed by the radical, two weeks or more after all acute symptoms had subsided. Occasionally, however, we did not wait long enough, so that some infection followed, especially in the plastic work. In three cases postaural fistulas resulted, and a plastic operation to close this had to be performed on one. None of the operators used electric or dental burrs, but these could probably have been used to a decided advantage. Sharp chisels, gouges and curettes were employed to remove the eburnized bone and facial ridge. While some of these radicals were performed before any attempt was made to get rid of polypi and granulations in the middle ear, I feel that it would be of distinct advantage to remove these several weeks before operation when possible and thus obtain a fairly healthy middle ear. This was the method followed in my cases, and it seemed to shorten the patient's stay in the hospital and prevent exuberant granulations from forming in the middle ear after operation. Wounds were dressed in accordance with the method of skin grafting that was used.

Skin Grafts.—There were thirty-eight with primary skin grafts; twelve with secondary; and six without graft. As will be seen from the above figures primary grafts were strongly favored. Secondary grafts were made for one of the following three reasons: First, to demonstrate the method of procedure; second, when an unusually badly infected mastoid made us

suspect that primary union would not follow a primary graft; and third, lack of time. The cases where no skin grafts were employed were those that developed some febrile disturbance, and it was either necessary to transfer them to some other institution or they could not be given an anesthetic with safety. In all these cases it was originally planned to do secondary grafts.

Of the two methods of primary grafting, that of using crepe de lile was the most popular. Twenty-seven, as against eleven, for the paraffin basket. When primary skin grafts were used one of the following two methods were in vogue:

First. *Crepe de lile*.—In this method a large skin graft was placed on crepe de lile and inserted through the postaural incision, efforts being made to carry the graft well down into the tubal portion of the cavity. This graft was held in place by iodoform tape, which after being so placed that pressure is evenly distributed all over the skin, was brought out through the external meatus and the postaural incision sutured. The iodoform tape and crepe de lile were removed in from five to seven days.

Second. *The Paraffin Basket*, as Devised by Dr. Harris P. Mosher.—This is made by placing in hot water previously prepared paraffin gauze which when softened can be put in the radical cavity postaurally and an accurate cast made of the entire cavity. This is then removed, superfluous margins trimmed off and the skin graft arranged over the basket so that it is completely covered. This is inserted through the postaural wound and packed with iodoform gauze which is brought out through the canal. The postaural incision is then sutured. The gauze filling the paraffin basket is removed in about four days without disturbing the basket, which is allowed to remain from seven to ten days.

The paraffin basket has the obvious advantage of getting the graft in close approximation to all parts of the radical cavity; it can remain undisturbed for a longer period than when the crepe de lile method is used and quicker epidermatization seemed to follow when it was successful. As to its disadvantages: If secondary infection follows the operation the wounds seemed to be unusually foul. It is rather difficult to remove the basket. At the best it is a rather painful procedure, and at the worst there is wounding of the external canal and loosening

of the skin flaps. While one frequently obtains beautiful results, and it unquestionably has many features to recommend it in selected cases, it never became an especially popular method on my service, although it was used almost exclusively on Dr. Mosher's. There is probably a certain knack in the successful use of this method of grafting. It has undoubtedly merit and is worthy of more general use. In a study of 100 radicals in the outpatient department my son found that 75 per cent of the cases in which the paraffin baskets were used had excellent cavities as compared with 50 per cent by the other epidermatizing methods.

When a secondary graft or no graft was used, the radical cavity was filled with iodoform tape and the postaural wound closed. This pack was usually left about a week. On removing it the cavity was filled with boric acid powder, when no skin graft was used, and the canal left open to give the radical cavity air and ventilation. If a secondary skin graft was to be used this was inserted directly after removing the iodoform pack. The skin was placed on crepe de lile and then arranged over a long, rather narrow plug made by folding three or four squares of gauze, each about four inches in size. This long, narrow plug, covered with the skin, is inserted through the canal and gently pressed into the radical cavity in such a manner as to permit the skin to come in contact with all portions of the cavity. At the end of three days the gauze and crepe de lile were removed and no further dressing used in the canal.

The End Results of the Skin Grafts.—As to length of stay in hospital, there was surprising uniformity. The average was as follows: Primary, with crepe de lile, 32 days; paraffin basket, 32.7 days; secondary, 34 days; no skin graft, 33 days. Skin graft complications were few. In four (that is, 15 per cent) of the primary crepe de lile cases there was slight infection in the postaural incision, requiring the removal of one or more sutures but not leaving a fistula or materially affecting the radical cavity or the end results. There were no complications in the secondary skin graft cases. One (or 14 per cent) of the cases without a skin graft became infected, and the sutures and packing were removed on the second day. A deep abscess in the neck had to be incised and drained. A secondary skin graft was done later and the end result was

satisfactory. Four (or 36 per cent) of the paraffin basket cases showed postaural infection. Two recovered by simply removing a suture, and the end results were excellent. The basket had to be removed early in the other two, one on the fourth and the other the fifth day, because of marked postaural infection. The skin over the radical cavity sloughed out so that both left the hospital with postaural openings. One of these, when seen a year later, had a postaural fistula that was still discharging and the radical cavity was so filled with granulations that a secondary operation was advised. The other case did better, the postaural fistula closed, but scar tissue had filled in the cavity above the facial ridge, thus dividing it into a small cavity with a minute opening in the antrum section and a fair sized middle ear section. When seen a year later the conditions were so satisfactory that no further operation was advised, but the patient was told that the ear should be carefully watched. From so small a number of paraffin basket grafts one should not draw unfavorable conclusions, as one might be inclined to do from this data. I do not think either of the cases were suitable for the basket; both probably had some acute condition so that any method would not have been satisfactory. If one suspects acute or subacute infection, it is best to plan on a secondary graft. One other rather amusing complication occurred in connection with the use of the paraffin basket. We were all questioning how long it was safe to allow it to remain. It was not unusual to leave it nine or ten days. As Dr. Mosher was anxious to have us find out, if possible, the maximum length of time it could be left, we let it remain in one case fourteen days with excellent results. Then we tried to leave it in another case for three weeks, but at the end of this time, when we attempted to remove it, the fibers of the basket had become imbedded in the epithelium of the radical cavity so that it was impossible to remove it. It pulled apart when grasped by forceps, so that it was necessary to give a general anesthetic and get it out piecemeal by a thorough curettement of the entire cavity. The ultimate result, however, was fairly good, but we never after this allowed the basket to remain longer than ten days.

Complete Epidermatization.—I was unable to determine this in most cases, as we usually let our patients go home as soon

as we felt that complications were not likely to develop, which was before complete epidermatization had taken place. It was usually about a month after the patients were discharged before the cavities were completely epidermatized. A few, however, would drag along for a much longer period. This point will be again referred to in the checkup of the cases.

The Flap.—The Koerner flap was employed in all the cases. Rubber tubing, split along one side, was used in the external portion of the canal in many of the cases. Roomier canals were obtained by this method than when we depended solely on the gauze in the canal to keep it dilated.

Sutures.—Mechel clips were used for closing the postaural incision in six. Silkworm gut in forty-three, black silk in one, chromacised catgut in six. It did not seem to make any special difference as to what sutures were used.

After Treatment.—This was dependent on the type of skin graft and the general condition of the wound. The packing was generally removed from the primary skin grafted cavities at about the end of the first week. From then on no further dressing was used over the external meatus. The radical cavity was left severely alone for the first two weeks. After this it was dried out and exuberant granulations if present removed. The nitrate of silver bead was used occasionally, but sparingly. Some of the operators filled the radical cavity with boric acid powder at the end of the second week. It always seemed to sweeten the cavity and favor rapid epidermatization. As the case progressed a saturated solution of boric acid in 70 per cent alcohol was used liberally in the wound, and when the patient was discharged he was instructed to use this freely at home. Scarlet red ointment was used occasionally and often seemed to be of decided benefit in helping to epidermatize some stubborn section of the cavity.

Stay in Hospital.—The average time was thirty-three days. A few were discharged at the end of the third week. The longest was nine weeks. While six were there seven weeks or over, most of these lived a long distance away or developed some complication necessitating this prolonged stay.

Complications.—In thirty-three of these fifty-four cases none were noted. Six had slight postaural infection. Severe infection in both the postaural wound and the radical cavity oc-

curred in four cases, requiring the removal of the sutures and the packing. There were four postoperative facial paralyses. One was due to trauma. It came on directly after the operation and cleared up within nine months. In the second and third cases it followed the secondary skin grafts and cleared up within a month. In the fourth it came on nine days after operation and lasted about three weeks. Four cases had preoperative facial paralysis. One of these followed an attempted radical by an outside specialist for a tubercular disease of the mastoid a year before admission and was probably permanent. We made an unsuccessful attempt to do an anastomosis between the facial and hypoglossal nerves. The facial paralysis in the other three cases was of short duration, and all cleared up within a few weeks after the radical operation. In one of these, a bilateral case with a large cholesteatome, the facial nerve was found exposed for about one inch on one side and one-fourth inch on the other. Severe vertigo for a week after the operation was noted in one case. One case developed marked atresia of the external canal. One had several severe hemorrhages from the edge of the Koerner flap and became quite exsanguinated. This hemorrhage usually came on during the night and was so severe that at one time we feared that the bone over the lateral sinus had become eroded and the sinus wall necrotic. Cauterizing the edge of the skin flap eventually controlled this unusual complication. One case developed erysipelas and another chickenpox. One case had meningism so severe that at first we thought we were dealing with a real meningitis. The temperature was elevated and the spinal fluid slightly cloudy with 1,500 cells but no organisms. On removing the sutures and packing, the case quieted down. One patient had convulsions, bronchopneumonia and erysipelas associated with a stiff neck and was desperately ill. A blood transfusion greatly benefited him and he ultimately made an excellent recovery. Sequestri comprising the cochlea and promontory were removed from two of the cases with preoperative facial paralysis. One of them (already referred to) had a permanent facial. The paralysis of the other cleared up within six months.

Final Checkup.—An attempt was made to check up on all these cases one or more years after the operation, and fairly satisfactory reports were obtained on forty-seven. Three lived

so far away that they were unable to return for an examination. Meager reports were obtained on four others. The radical cavity was dry in twenty-five and but slightly moist in fourteen. Twelve contained granulations and offensive secretion. Two of these latter were considered failures and both needed another radical. The others should become dry on receiving appropriate treatment, as all had reached this condition through neglect. The fourteen moist ears had, on the whole, good cavities but were moist either from neglect or from some acute flareup in the nasopharynx. Four cases were readmitted some weeks after leaving the hospital, for infection about the auricle or in the radical cavity. The infection in two of these subsided under local treatment. The third case, which was taken home against advice, had when readmitted a red and swollen auricle, but this subsided under treatment after the removal of a large bunch of granulation tissue which had blocked the canal and interfered with the drainage and ventilation of the radical cavity. The fourth case, when admitted two months after his discharge, and on another service, was suffering severe pain through the eye of the affected side and had a blurring of his vision. There was infection in and about the radical cavity, and organized granulations extended across the facial ridge, probably producing obstruction to the drainage of the posterior portion of the radical cavity. A brain abscess was suspected, and Dr. Cahill did an exploratory operation on the middle fossa but nothing was found. There was marked improvement for three weeks, then the pain returned, and Dr. Meltzer explored both the temporal and cerebellar regions of the brain but found nothing. Blood cultures and spinal fluid were both negative. There was some blurring of the nasal side of both optic discs. The patient died an hour after the exploratory operation. While death was attributed to basilar meningitis there is some question as to the possibility of its being an infection of the lateral sinus. Dr. Ayer, our neurologist, saw the case and reported as follows: "Patient mentally alert. No aphasia or speech defect noted. Meningeal symptoms lacking—i. e., no stiff neck. Complete paralysis left Vi; hyperesthesia and hyperalgesia left V (first and second divisions); absence of motor III, V and VII palsy. Extremities normal. Gradenigo's syndrome left. Advise exploratory operation."

Dr. Ayer also did a manometer test and found that the jugular on the affected side did not respond quite as well as that on the unaffected, 310 against 390. The spinal fluid was turbid but contained no growths. Dr. Ayer thought there was increased intracranial pressure and believed that the evidence pointed to a lesion below the tentorium, probably an abscess on the left side, but he added that thrombosis of the left lateral sinus was a possibility. This, as far as we know, has been the only fatality since the cases were discharged.

Records were made on seventeen of the outpatients cards that exuberant granulations had been removed and the bases cauterized. This treatment was probably given to a much larger number, as nearly every radical case occasionally needs it. One patient developed nausea and vomiting two and one-half years after her discharge and was admitted to the hospital for a week. These symptoms rapidly disappeared as soon as the patient was put to bed. A caloric test failed to develop any response on the affected side after five minutes' douching. Three of the radical cavities, through neglect, became so filled with debris that it was necessary to readmit these cases and clean out their cavities under general anesthesia. When once clean they all did well. Another case developed pernicious anemia after leaving the hospital but improved rapidly under raw liver diet.

The follow-up work was most instructive. Practically every case that reported regularly had a good dry cavity, but many of the patients failed to so report after the ear was once dry. Eventually the accumulated desquamative epithelium became moist, the epidermis melted away, and when they finally turned up they had red moist cavities with circumscribed areas of exuberant granulations. As this is a condition that occurs through neglect, some systematic effort should be made to follow them up. The Social Service should look up every case that fails to report to the outpatient at least twice a year, and oftener if the ear is moist. In private practice our radical cavities are easily kept dry by such attention. I have urged the Social Service of the Massachusetts Eye and Ear Infirmary to institute some such follow-up system and hope that this will soon be done. There also may be need for more intelligent care of these cases in some of our clinics. Men are often work-

ing in these clinics who from lack of training do not always appreciate the importance of following up these cases, so it would seem advisable to have these radicals passed on by some senior man who has had experience in operating on them, when they report back to the clinic. After spending so much time on these cases in the hospital it is of the utmost importance that they should not come to grief after they leave the house service through avoidable neglect or faulty methods of after treatment.

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SUMMARY.

A study was made of fifty-four radical mastoid cases which were operated upon at the Massachusetts Eye and Ear Infirmary. Various tests and examinations were tabulated and previous treatment and operations noted.

A third of these cases had been previously operated upon once or more for mastoiditis, and about half had previously had their tonsils and adenoids removed.

The nose was practically negative in 75 per cent of these cases. Half of them had cholesteatome and most of them had been under treatment in the outpatient department for some years.

The fundi and visual fields were practically negative in all the cases.

The Koerner flap was the only one used.

Primary skin grafts were usually employed. Of the two methods, crepe de lile and paraffin basket, the former was given the preference in the ratio of 27 to 11. The end results were about the same, irrespective of the method of epidermatization.

Hearing was improved in five cases, unchanged in six and worse in seventeen. It appeared to fail for some months after the operation, probably due to cicatricial tissue about the stapes.

The average stay in the hospital was thirty-three days. There were several severe postoperative complications. The only case of traumatic postoperative facial paralysis cleared up at the end of nine months. The only fatality occurred from a reinfection about the mastoid three months after the patient's discharge from the hospital. Two of the radicals were failures and secondary operations advised.

The follow-up work clearly demonstrated that these cases do not receive proper attention after their discharge from the

hospital. Practically all the cases that returned regularly to the outpatient had good cavities and dry ears. Many only returned when the discharge from their radical cavities became offensive. Some had to be readmitted for the removal of granulations and débris under a general anesthetic. Others had red cavities, largely denuded of epithelium, and containing islands of granulations, but recovered under appropriate outpatient treatment.

With systematic follow-up work on the part of the Social Service these postoperative complications should be avoided. The hospital treatment of these cases was satisfactory, while the after care was, on the whole, unsatisfactory. Recognizing that this is so, appropriate steps to correct it should at once be taken.

I am happy to state that since the completion of this paper and its perusal by Miss Amy Smith, the head of the Social Service Department of the Massachusetts Eye and Ear Infirmary, the following method of following the radical mastoid cases has been instituted:

First.—The outpatient Social Service worker of the Ear Department gets in touch with all the present radical mastoid patients while they are in the hospital. She thus becomes acquainted with them and gains their confidence.

Second.—When these cases are discharged she puts them on her follow-up list. The outpatient records are marked so that when the patients return they are referred to her and she takes them to one of the senior men in the clinic, and, after they have been treated, notes the date when they should return. If the case does not report on time, a note is sent, and if this brings no response the patient receives a visit from the Social Service worker. By this follow-up method it is hoped that permanent contact with the patient will be maintained. Twice a year, or oftener if necessary, these patients are to be brought to the outpatient for inspection of their radical cavities.

Third.—Lists are being made of the previous radical patients and notices sent to them to report to the outpatient, so that it is hoped that contact will soon be made with most of these old cases and a semiannual checkup established.

XXXIV.

MAGNETIC AID IN THE BRONCHOSCOPIC AND ESOPHAGOSCOPIC REMOVAL OF FOREIGN BODIES FROM THE AIR AND FOOD PASSAGES.*

BY CHEVALIER JACKSON, M. D., Sc. D., LL. D., F. A. C. S.,
PHILADELPHIA.

Every now and then there comes a letter inquiring about magnetic extraction of a foreign body from the lung. Sometimes it is a letter expressing surprise that this means has not been developed to a greater extent; at other times it is a letter with what its author deemed constructive suggestions. The last letter was from an electrical engineer who proposed that we should develop the field together and divide the profits which he imagined would accrue from patent rights to be obtained!!!

Any attempt to follow the brilliant achievements of the late William M. Sweet and other ophthalmologists with the magnet will soon reveal the fact that while magnetism can be of occasional use in extraction of foreign bodies from the air and food passages, there are certain limitations to its general usefulness for the peroral extraction of foreign bodies.

The limitations to the practical utility of magnetism in bronchoscopy were pointed out over twenty years ago; and they remain about the same today. The following points are of importance in considering the problems of magnetic aid in the peroral removal of foreign bodies from the air and food passages.

1. The foreign body must be of iron or steel or cobalt, wholly or partly. This cuts out the use of magnetism in over ninety per cent of the cases. Only a fraction of the nearly two thousand foreign bodies in the collection of the Bronchoscopic Clinic at the College of Physicians of Philadelphia are magnetic.

*Read before the American Bronchoscopic Society at Atlantic City May 21, 1927.

2. The foreign body must be free to be moved.
3. The attraction of the magnet for the foreign body is no greater than the attraction of the foreign body for the magnet; a small foreign body pulls but slightly on the magnet and *vice versa*. Hence,
4. The clinical efficiency of increasing the size and power of the magnet is limited by the fact that we cannot increase the size of the foreign body. If the foreign body were a cast-iron dumbbell a powerful electromagnet would pull it out through the chest wall; and the patient could be suspended in the air by magnetic power.
5. For the reasons given, the probabilities of the usefulness of magnetism in the removal of a foreign body from the air or food passages are inversely as the size of the foreign body.
6. The magnetic force in practical use diminishes even more than directly as the square of the distance; hence
7. The location of the foreign body must be such that, if a core magnet is to be used, the core can be brought within reasonable distance of the foreign body.
8. Prolongation of the core lessens only to a small degree the loss as the square of the distance, but to a limited extent it may be useful if it enables contact.

The fundamental limitation, of course, is that there are only three common substances that are susceptible to magnetic attraction, namely, iron (or steel), nickel, and cobalt. The foreign body must, therefore, be composed partly or wholly of these for magnetic aid to receive any consideration in the solution of the problem of extraction. Some alloys of iron are not magnetic and there is a nonmagnetic steel and a nonmagnetic cast-iron. Certain nickel-iron alloys can exist in either of two states, magnetic and nonmagnetic, according to the heat-treatment they have received. On the other hand, certain alloys of manganese, copper and aluminum may be magnetic. Perhaps the most notable of the recent magnetic discoveries is the nickel-iron alloy known as permalloy, which has such a high degree of permeability that, in weak magnetic fields or under weak magnetic forces, it can attain a magnetization hundreds of times greater than pure iron or steel. Permalloy, which has been extensively used in telephonic and telegraphic work, has not yet been encountered, so far as I know, as a foreign body.

But in view of the case herein reported it becomes interesting to determine to what extent magnetic permeability may be increased in steel jacketed bullets, which are far from being infrequent as foreign bodies.

INSTRUMENTARIUM FOR MAGNETIC EXTRACTION OF FOREIGN BODIES FROM THE AIR AND FOOD PASSAGES.

An extensive series of experiments were carried out twenty years ago, resulting in the development of the magnetic means listed below.

1. Magnets of various shapes and sizes.
2. Rod magnets.
3. Rods for permanent magnetic activation.
4. Rods for interrupted magnetic activation.
5. Vertebrated rods for magnetic activation.
6. Chains for magnetic activation.
7. Magnetic forceps.
8. Forceps for interrupted magnetic activation.
9. Projected core magnets.
10. Solenoid magnets.
11. Powerful magnet (Sweet or Haab) for activation of rods, forceps, chains, etc.

Electromagnets of rodlike form for insertion through the bronchoscope are not very powerful, because of the small number of turns of wire possible within the limits of diameter of the tracheobronchial lumen.

Permanent rod magnets must be of glass-hard steel, "hardened right out," as the toolmakers say, in order to retain their magnetization. The cobalt steel of Honda is the best for rod magnets.

Rods for interrupted magnetic activation are made of soft iron, rounded at the distal ends. They may be energized at will by bringing the end of the core of a powerful magnet, preferably Sweet's eye magnet, against the proximal end. If it is intended that complete demagnetization take place as soon as the contact is broken, or the current in the magnet is broken, it is essential that the rod be of soft iron. Steel will retain more or less of its polarity after interruption of the magnetic force, and hard-tempered tool steel becomes more or less permanently magnetized.

Vertebrated rods are similar to those described in the foregoing paragraph, with the added advantage of a few centimeters vertebrated distal end. The entire rod may be vertebrated if desired.

Magnetic forceps are of two kinds. They may be permanently magnetized or may be energized at will by the contact of a powerful electromagnet applied at will to the proximal end. This gives more attractive power than is possible in a permanently magnetized forceps. For maximum efficiency the forceps and cannula should both be of steel and should be as heavily constructed as may be possible for use in the particular case. It may in some cases be advisable to dispense with the bronchoscope, so that a heavier instrument can be used; but this will very rarely be the case, because such a heavy forceps would not enter the small bronchi and would, moreover, require close watching to see that the bypassage for air was sufficient. The usefulness of magnetic forceps is limited.

Projected core magnets are more powerful than rod magnets but are rather unwieldy.

Solenoid magnets were very promising experimentally, but probably their clinical usefulness is very limited, for the reasons given above.

Magnetic Instrumentation.—A few points must be borne in mind.

(a) Diameter. If the magnetic device is a close fit in the larynx or trachea, about one minute is as long as it can be kept in the windpipe without risk of asphyxia. An instrument of the diameter of either main bronchus would probably allow a bypassage of air in trachea. A close fit inside of a close fitting bronchoscope would be considered as occluding the airway completely. Hollow magnets give an airway but have no advantage over the same amount of metal in a solid rod of proportionately less diameter. In working it should always be kept in mind that a magnetic foreign body may become polarized under the influence of proximity to or in contact with a magnet. Therefore, polarity of instruments must not be reversed, because like poles repel; the result of reversal would be repulsion, not attraction.

Any form of magnet exerts its greatest power on contact. Therefore, contact of the magnet with the foreign body should be made if possible. Out of contact, the loss of power for all practical purposes, is at a greater rate than as the square of the distance. As there is no substance that will impede magnetic lines of force, intervening tissues do not prevent magnetic attraction except as they may hinder movement of the foreign body. It is the intervening distance and not the intervening tissues that lessens practical efficiency of the magnet for our purposes.

Magnetic Fixation.—In a recent case, elsewhere reported, a new method of using magnetism as an aid to bronchoscopy was developed. A steel jacketed bullet, free to move in an abscess cavity and located "up around the corner" in the left upper lobe, was drawn down and fixed in the abscess cavity by a large, powerful eye magnet applied at the proper position on the external chest wall. The fixation enabled me to get the forceps securely closed on the bullet in the precise endwise position necessary for removal through the tightly strictured fistula communicating with the cavity. The bullet could not have been withdrawn through the mouth by any magnet that could be inserted through the natural passages. It might also be added that had the bullet been altogether of lead it could not have been moved at all by any magnet.

XXXV.

GLOSSOPHARYNGEAL NEURALGIA: A REPORT
OF FIVE CASES.

BY FRENCH K. HANSEL, M. D., M. S.,

ST. LOUIS.

Since Fothergill's classic description of trigeminal neuralgia, about 150 years ago, many hundreds of cases have been reported in the literature. The disease has been considered a definite clinical entity but no definite etiology has been discovered; therefore, it is considered essential or idiopathic. Until only a few years ago a similar idiopathic neuralgia affecting the glossopharyngeal nerve was unknown, but now we know that such a disease does exist.

This presentation on glossopharyngeal neuralgia is being made¹ with the object of adding five cases to those already reported in the literature, and² with the object of pointing out certain features of this neuralgia which are important to the otolaryngologist.

Glossopharyngeal neuralgia is an affection manifested by the occurrence of paroxysmal attacks of sharp, excruciating and lancinating pains in the tonsillar region, base of the tongue and lateral wall of the pharynx, radiating to the ear and surrounding region. In all its characteristics the pain is similar to that of trigeminal neuralgia, differing only in the area of distribution.

The individual attacks last only a few seconds and are chiefly excited by talking, chewing and swallowing. An attack may be excited by pulling the tongue, as in making a laryngoscopic examination, or by tickling the tonsillar region or lateral wall of the pharynx. A trigger area may therefore be found in these regions. If a trigger area is present, cocainization of these areas may make it difficult to excite paroxysms of pain. The periods of pain and freedom from pain may vary from weeks to months and sometimes years, but always recur.

REVIEW OF LITERATURE.

In the following review of the literature I have chosen for discussion only those reports on true or idiopathic glossopharyngeal neuralgia. The publications on neuralgic-like disturbances of the glossopharyngeal nerve will be considered in the general discussion at the close of this presentation.

The first report in the literature on idiopathic glossopharyngeal neuralgia was that of Sicard and Robineau,¹ in 1920. They reported three cases of what was termed "Algie velo-pharyngee essentielle." In each case the pain was paroxysmal, sharp and excruciating, induced by swallowing, chewing and speaking. The pain was referred to one side of the pharynx, tonsil and soft palate and radiated to the region of the ear. Since the pain was of long duration and there was no organic lesion to account for it, the condition was considered as idiopathic or essential. Treatment consisted in the section of the glossopharyngeal nerve and pharyngeal branches of the vagus in the neck and the superior cervical ganglion. In all cases the pain was relieved.

In 1921, Harris² reported two cases of typical glossopharyngeal neuralgia. In one case, the third division of the fifth nerve was injected without relieving the pain.

In 1923, Doyle³ of the Mayo Clinic reported four typical cases. One patient had an operation for trigeminal neuralgia without relief. Later the glossopharyngeal nerve was avulsed and the pharyngeal branch of the vagus cut with relief. In another case, the third division of the fifth nerve and the auriculotemporal nerve were injected without results. In the other two cases no treatment was administered.

Shortly after Doyle's report, H. I. Lillie⁴ of the Mayo Clinic presented a treatise on glossopharyngeal neuralgia from the standpoint of the otolaryngologist. He emphasized the importance of differentiating glossopharyngeal neuralgia from nasal ganglion syndromes and trigeminal neuralgia. He reviewed three cases previously reported by Doyle. In the discussion of Lillie's paper, R. O. Barlow reported one case in which avulsion of the ninth nerve was performed with satisfactory results.

In 1924, Adson⁵ of the Mayo Clinic reported four cases, two of which had previously been included in Doyle's publication. In one of Adson's cases peripheral avulsion was attempted without success, but in two cases the glossopharyngeal nerve and the pharyngeal branches of the vagus were cut with relief of symptoms. Adson describes the technic of peripheral avulsion and has perfected an intracranial operation on the cadaver which he believes superior to the external operation.

In 1926, Singleton⁶ reported two cases. In one instance the patient was unsuccessfully operated on for trigeminal neuralgia, but later a diagnosis of glossopharyngeal neuralgia was made. In the second case, the patient was relieved of all symptoms by avulsion of the ninth nerve.

In 1926, Miller⁷ reported a typical case, but no treatment was administered. A few months later Albright⁸ reported another case. Many attempts were made to relieve the pain, such as tonsillectomy, extraction of teeth and injection of the fifth nerve, but failed.

In Harris' text on "Neuritis and Neuralgia" (1926), he refers to a third case of glossopharyngeal neuralgia, two having been already reported in 1921. The disease lasted for a period of fifteen years. There were long periods of freedom from pain but for some time before the patient died of another disease the attacks were almost continuous.

In 1926, Goodyear¹⁰ added another case having all the characteristic features. The pain radiated to the region anterior to the ear, and a definite trigger area was present on the posterior pillar.

A most comprehensive article on this subject has recently been published by Dandy.¹¹ He reviewed the literature thoroughly, reported two cases and was the first to report a successful use of the intracranial operation. Following the operation upon his two patients he was able to demonstrate the nerve supply of the glossopharyngeal nerve and has contributed some valuable information on this phase of the subject. In this review of the literature, covering a period of seven years, twenty cases of glossopharyngeal neuralgia have been reported. With the addition of the five cases herein reported, the total is brought to twenty-five. As the disease becomes more generally known, many cases should soon accumulate.

CLINICAL FEATURES.

Although Dandy has most comprehensively summarized the clinical features of glossopharyngeal neuralgia, it may be well to repeat them to emphasize the characteristics of this neuralgia. In reviewing the twenty cases reported in the literature and the five cases appended here, a striking uniformity of symptoms is noted. The paroxysms of pain always start in some part of the terminal distribution of the glossopharyngeal endings—the pharynx, tonsil or base of tongue.

The pain was unilateral in all cases; no bilateral distribution has been reported. The pain is induced by swallowing, talking or chewing. Swallowing cold liquids seems to be the most effective in exciting attacks. The pain is always excruciating, sharp and lancinating. The individual attacks last only a few seconds, rarely more than a minute. The attacks usually come on in series, lasting from a few days to a few weeks. Long or short periods of freedom from pain usually occur. There may be an absence of paroxysms for years, but when the pain returns the same area becomes affected. In a few of the cases the pain started originally in the ear, the lobule of the ear or at the angle of the jaw, but the attacks were induced by swallowing, chewing, etc. The pain starting in the pharynx, tonsil or base of the tongue radiates to the ear or surrounding region, such as the lobule, meatus, concha or in front or behind the ear. It is probable that the pain reaches the ear through Jacobson's nerve, but since the glossopharyngeal nerve has no sensory distribution in the pain areas about the ear we must assume that it is referred through the terminal branches of the trigeminal and vagus in this region. The fifth, ninth and tenth nerves are intimately connected in the region of the ear. The external ear may be sensitive to touch, and paroxysms of pain may be induced from this location.

This was true in two of Doyle's cases, one of Adson's and one of Harris'. Besides the chief method of precipitating attacks—the drinking of cold liquids—other exciting causes are: Swallowing (mostly liquids), talking, yawning, coughing, sneezing, touching the ear, turning the head or touching the angle of the mandible. Attacks may also occur spontaneously. A trigger zone was present in three of the cases reported here

(base of the tongue), in both of Dandy's cases, in one of Adson's, Goodyear's case and Albright's case.

Pressing down on the tongue with a spatula seems to be the most effective in producing a trigger action.

GLOSSOPHARYNGEAL NERVE SUPPLY.

In connection with the intracranial section of the glossopharyngeal nerve, performed by Dandy for the relief of glossopharyngeal neuralgia, he tested out the sensory supply of the nerve and has given a detailed description of it. No previous method of testing out the nerve supply has been reliable. Attempts have been made in connection with paralysis of the glossopharyngeal nerve, associated with brain tumors, extracranial tumors and fractures of the base of the skull, but on account of the close relationship and connection with other cranial nerves, satisfactory information has never been obtained. Therefore we are indebted to Dandy for his excellent contribution to our knowledge of the sensory distribution of the glossopharyngeal nerve.

The after effects of division of the glossopharyngeal nerve intracranially were almost the same in both of Dandy's cases. They were as follows:

Motor Changes.—Symptomatically there were no objective or subjective disturbances. Swallowing and constrictor action of the pharynx were unaffected. The soft palate moved normally. The stylopharyngeus muscle, supplied by the glossopharyngeal nerve, could not be tested for loss of function.

Alterations of Taste.—In both cases there was a complete loss of taste on the posterior third of the tongue. This confirms the usual conception of the nerve supply of taste for that part of the tongue, but in the other cases there was a slight but definite diminution. Dandy states that further details on this aspect of the subject will soon be reported by Dr. Dean Lewis.

Sensory Changes.—The sensory changes were illustrated by sketches. "The only differences noted in the two cases were that in one patient sensation was perceived in the eustachian tube for a few millimeters below the pharyngeal orifice; in the other, there was no sensation for a distance of about 2.5 centimeters. In one patient, the sensory deadline exactly bisected the uvula; in the other, almost the entire uvula was anes-

thetic. In both patients a cup of sensation remained at the vault of the nasopharynx, doubtless being supplied by the trigeminal nerve. The remainder of the pharynx, anterior, posterior and lateral, down to the epiglottis (including the posterior aspect), the vellicula and the pyriform sinus was anesthetic. Anteriorly and posteriorly, the line of anesth sia ended sharply at the midline. The soft palate is supplied by the glossopharyngeal nerve only in a narrow rim at its oral surface and over a greater extent on its nasal surface. The tonsil and eustachian orifice were insensitive in both cases.

The epiglottis was anesthetic on its posterior aspect: the line of demarcation of the area with the normal sensation of the anterior surface from that supplied by the vagus is a sharp line along the rim of the epiglottis. The precise line of demarcation between the area of normal and of lost sensation was difficult to determine in two places—above, at the vault of the nasopharynx, and below, at the beginning of the esophagus. There was no area of anesthesia in the nasal cavity."

Secretory and Sympathetic Nerves.—There was no disturbance of salivary secretion. There was no disturbance which could be attributed to the small superficial petrosal nerve, which is a continuation of Jacobson's nerve through the tympanic plexus.

DISCUSSION.

The diagnosis of idiopathic glossopharyngeal neuralgia is made entirely from the symptomatology and the observation of the attacks. The location of the pain distinguishes it from trigeminal neuralgia, although the type of pain is identical. In a great many of the cases reported, the disturbance was erroneously diagnosed as trigeminal neuralgia and treated as such. In some instances no definite diagnosis was made; tonsil and nasal operations were performed to relieve pain but without success.

Tumors involving any part of the distribution of the glossopharyngeal nerve have been known to cause paroxysms of pain somewhat similar to the idiopathic type of neuralgia. The pain, however, is apt to be more constant, less paroxysmal and not subject to long periods of remission. As a rule, there are sensory changes over the distribution of the ninth nerve.

In 1910, Weisenburg¹² was the first to report a case in which the pain was localized in the distribution glossopharyngeal nerve; the symptoms closely resembled those of glossopharyngeal neuralgia. The patient had a cerebellopontine angle tumor, with symptoms of irritation of the fifth, ninth and twelfth cranial nerves. The symptoms began with pain in the lower jaw, teeth, nose, eye and forehead. He had injections and peripheral avulsion of the branches of the fifth nerve without relief. Four years later there was a paralysis of the superior oblique and superior rectus muscles of the eye. A gasserian ganglion operation was performed without relief of pain. Later the pain extended to the throat. The posterior auricular nerve was cut and the lesser occipital nerve injected with only slight relief.

About a year after the gasserian operation the symptoms became worse. He complained of sharp stinging pains, which began in the back of the right side of the tongue and extended to the ear and down the side of the throat, at times extending to the shoulder. These would last for a few minutes and were usually succeeded by a burning sensation in the parts. He had also a constant burning sensation in his tongue and throat, and sensations as if roaches were crawling over these parts, and his throat was constantly dry. Touching the pharynx and tongue would bring on attacks of pain. Swallowing always brought them on. About five years after the onset he began to have involuntary movements of the tongue. The patient finally died, six years after the onset of the pain.

At autopsy a tumor was found at the right cerebellopontine angle, lying directly on the sensory and motor roots of the fifth nerve. The ninth and tenth nerves were stretched. It was impossible to tell whether the twelfth nerve had also been stretched. Microscopic examination of the cranial nerves showed the third, fourth, sixth, seventh, eighth and twelfth slightly degenerated. The ninth and eleventh nerves were normal. The optic was normal.

I have described Weisenburg's case in detail to emphasize some of the points in differential diagnosis. The persistence of pain in the distribution of the fifth nerve after operation on the gasserian ganglion should have suggested a more central

irritation. While the pain over the distribution of the glossopharyngeal nerve somewhat resembled that of glossopharyngeal neuralgia late in the course of the disease, the sensory disturbances in the pharynx were indicative of irritation of the ninth nerve. Sensory disturbances are never present in true glossopharyngeal or trigeminal neuralgia. In cases of tumor of the gasserian ganglion, the sensory disturbances of pain, touch and temperature are very characteristic.

In Sheldon's¹³ report of four cases of tumor of the gasserian ganglion, he stated that sudden paroxysms of pain were infrequent and in no instance were they influenced by eating, speaking or external irritation. Malignant tumors of the nasopharynx may also involve the branches of the fifth nerve and cause symptoms identical with those of tumor of the gasserian ganglion.

In Harris' book⁹ on Neuritis and Neuralgia, he describes several cases in which there were paroxysms of pain in the throat and ear as a result of malignancy of the tonsil, aryepiglottic fold or palatoglossal fold. The author observed a case of abscess of the tongue which came on with paroxysms of pain in the throat and ear similar to glossopharyngeal neuralgia.

In differentiating the various pains about the throat and ear it is important to remember that several cranial nerves intermingle in this region. Irritation of one nerve may cause pain in some part of the distribution of the other. Harris states that the nerve supply of the external ear is very complex. In addition to the posterior auricular branches from the second and third cervical nerves and branches from the sympathetics, no fewer than four cranial nerves give branches to it: (1) the auriculotemporal branch of the third division of the fifth; (2) sensory branches from the geniculate ganglion of the facial; (3) Jacobson's nerve from the ninth, and (4) Arnold's nerve from the tenth or vagus nerve.

Harris describes a case of chronic paroxysmal neuralgia of the superior laryngeal nerve. The pain started in the region of the middle of the posterior edge of the thyroid cartilage and radiated to the ear. The pain was not excited by swallowing but could be brought on by excessive talking. There was no pain in the throat.

Pains in the throat and ear, but quite unlike paroxysmal tic, are known to occur in cases of tuberculosis and cancer of the larynx and epiglottis.

Harris also speaks of auricular neuralgia. Severe paroxysms of pain in the ear are excited by rubbing or touching the auricle.

In typical cases the syndrome of glossopharyngeal neuralgia is so classical that a diagnosis can be made with certainty, but in atypical cases some organic lesion should be sought as a cause.

By employing the intracranial operation for the relief of glossopharyngeal neuralgia, as advocated by Dandy, the cranial nerves may be explored at the medulla. If a tumor is present, it may be removed by this operation.

Case 1.—A woman, aged 23 years, entered Barnes Hospital January 29, 1917, complaining of attacks of sharp, excruciating and lancinating pain in the left side of the pharynx, radiating to the left ear. The attacks had been occurring intermittently for a period of two years, every four or five days.

The pain lasted only a few seconds and was brought on by chewing, swallowing and talking. The attacks could also be induced by pressing down on the tongue with a spatula.

General examination and special examination of the ear, nose and throat were negative. The patient was advised to have the wisdom teeth extracted. This was done but there was no relief of pain.

The character and location of the pain leave no doubt as to the diagnosis of glossopharyngeal neuralgia in this case.

Case 2.—A woman, age 47 years, entered Barnes Hospital October 23, 1924, complaining of paroxysmal attacks of sharp, excruciating pain the right side of the pharynx, radiating to the right ear. The attacks appeared suddenly and lasted for a period of thirty to sixty seconds. They were brought on by eating and talking. During a period of five years the patient had had eight attacks of pain, each lasting several weeks. During the interims she was free of pain.

During the attacks the pain was so excruciating that the patient refused to eat. When the pain appeared she would grasp something with the hands while enduring the suffering. If she

happened to be eating during the time, she could neither swallow nor expectorate the food.

The examination of the ears, nose and throat was negative. Tests made for the sense of taste revealed nothing abnormal. The general and neurologic examinations were also negative.

The symptomatology and location of the pain are typical of glossopharyngeal neuralgia.

Case 3.—Mrs. R. F., a woman, age 61 years, entered Barnes Hospital July 13, 1924, complaining of attacks of sharp, severe and lancinating pains in the right side of the throat, radiating to the right ear. The first attacks began in February, 1923, and continued for a period of six weeks. They appeared again in July, 1923, and lasted until October, 1923. When she entered the hospital, the attacks had been intermittent for a period of six months. The individual attacks lasted only a few seconds and were brought on by chewing and swallowing. Drinking cold water was particularly effective in inducing the pain. The patient had had three nasal operations for relief of the pain without results.

During the attacks the patient appeared to be suffering the most agonizing pain. She grasped the right side of the neck and ear, just as a patient grasps the face when suffering with trifacial neuralgia. The general, neurologic, special and laboratory examinations were negative. An X-ray of the cervical spine showed hypertrophic arthritis.

A diagnosis of glossopharyngeal neuralgia was made, but the patient refused operation.

Case 4.—J. L., a man, age 52 years, was seen at Barnes Hospital September 29th, 1924. He gave a history of having suffered with attacks of sharp, excruciating and lancinating pain in the left side of the throat and neck, radiating to the left ear, intermittently for a period of ten years. The individual attacks lasted about thirty seconds and were brought on by talking, chewing or swallowing. Four attacks or periods of pain, lasting one to three weeks, occurred during the ten years.

The examination of the ear, nose and throat showed nothing to account for the pain. The patient had been operated upon for lung abscess and was suffering with diabetes, but these conditions had nothing to do with the neuralgia.

A diagnosis of glossopharyngeal neuralgia was made, but treatment was not advised on account of the patient's poor condition.

Case 5.—T. W., a man, aged 60 years, was seen on August 3, 1927, complaining of attacks of sharp, stabbing and excruciating pain in the left side of the pharynx, radiating to the left ear. The attacks always appeared suddenly, lasting for a few seconds, and then disappeared. The duration of the neuralgia was about two years. During this time a number of these attacks appeared, but there were long periods of several months when he was entirely free of pain. The pain on the external ear was referred to the region of the tragus. The attacks were brought on by swallowing, chewing and especially talking.

Examination of the nose, throat and ears was negative. No trigger area could be demonstrated. The symptoms in this case definitely indicated that the patient was suffering with glossopharyngeal neuralgia. Periods of pain were of short duration, and since the patient had not had many attacks he did not wish to have any form of treatment.

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XXXVI.

GROSS AND MICROSCOPIC PATHOLOGY IN TWENTY-THREE CONSECUTIVE EXTERNAL
FRONTAL ETHMOSPHENOID
OPERATIONS.

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As indicated by the subject, this paper is naturally divided into two parts: the first deals with pathology as seen at operation, the second with the microscopic findings in the bone and soft tissue. Some of these cases, which were clinically chronic, were found to present microscopic evidence of acute inflammation, and, vice versa, in studying the one acute case presented, there was as much evidence of chronic pathology as was found in the true chronic cases. We feel that the study of other similar cases seen by us verifies this statement. Some acute processes, then, are only acute exacerbations of a chronic condition.

While inflammation of the paranasal sinuses has been known and commented upon since the early history of medicine, the most systematic study of this condition, until the past few years, has been by our German confreres. Recently much work has been done by the fellows of this society. Notable among these is Joseph C. Beck, who wrote concerning acute sinus disease as follows: "The overlying skin and periosteum are edematous and acutely infiltrated; the bone bleeds freely, and as soon as the cavity is opened a flow of secretion escapes under tension. The mucous membrane lining of the cavity is markedly thickened and edematous; it bulges through the opening and bleeds very freely and appears to be lifted off the underlying bone. Microscopic examination shows marked thickening of the subepithelial structures. There is marked engorgement, and there are some thrombotic vessels. The

underlying bone shows an acute osteitis and in places may show definite bony necrosis. This is of particular importance in relation to secondary and cranial infections."

Zuckerkindl, one of the early writers on this subject, classified the clinical findings as osteoma, cysts, cholesteatoma, polypi, necrosis, periostitis and mucous membrane tumor.

Gerber, in discussing cholesteatoma of the frontal sinus, stated that there was practically always a history of previous opening from the skin surface into the sinus.

Oppikofer investigated the mucous membrane of one hundred cases of chronic suppurative sinus disease. In two frontal cases there were extensive metaplasia changes from cylindrical to pavement epithelium. These were cases of long duration. He found flat epithelium in twenty-seven of sixty-six antrum cases, seven of twenty-two frontal cases, one of ten ethmoid cases, and none in two sphenoid cases, but he saw no reason why it should not occur in the sphenoid. In sixty-five cases of acute sinus disease he did not find metaplasia. He considered any case chronic in which metaplasia was present. Manasse said: "Metaplasia of epithelium as described by Oppikofer, and Froning, I have not seen in chronic sinus suppuration." Skillern spoke of metamorphosis of ciliated into pavement epithelium as a rather common occurrence.

Oscar Beck found Herzog's histologic study of the ethmoids in four cases of retrobulbar neuritis interesting. In Herzog's cases there was very little found clinically. In these cases, however, the findings were histologically similar: hyperplasia of the mucosa with infiltration of round cells and serous infiltration of the bone marrow. Beck also reported four cases of retrobulbar neuritis in all of which he found histologically edema of the ethmoids of more or less marked degree, the occurrence of plasma cells, and eosinophiles. He stated that both of the latter are found only in pathologic conditions. Hyaline bodies were seen in one of his cases but he considered that to be of comparative rarity. He found neither bone pathology nor positive evidence of purulent inflammation.

Paul Manasse found the bony changes in acute and chronic sinus disease in a manner much the same, the acute process usually becoming a chronic one. These changes were principally collections of osteoblasts to form osteoid or new formed bone deficient in calcium salts, granulation tissue and later, fibroblasts. He found the same process playing upon the dural surface of the bone as on the surface facing the sinus mucosa. This formation of osteoid or new formation of bone ran concurrently with connective tissue formation and resorption of bone as evidenced by enlargement of the marrow spaces. He rarely found osteoclasts and when found these cells were in the marrow spaces. He found bone atrophy, but did not find bone thickening. In describing the microscopic findings in the acute cases, he says there is usually no defect in the epithelium, but that the increase in thickness is due to accumulation in the subepithelial layers. Manasse classified chronic suppurative paranasal sinus disease microscopically into (a), edematous or dropsical; (b) granular or infiltrating; (c) fibrous; (d) a mixture of any two or all of these forms. He found connective tissue change common and usually much thickening in the subepithelial layer. This increase in cellular structure was made up of spiral, round, starformed, plasma, eosinophile and pigment cells.

Benson, in a recent personal communication, wrote as follows: "The epithelium gives rise to glands of two types, mucous and serous, and it appears from my studies that the Schneiderian epithelium itself undergoes, under varying conditions, a transition to the mucous or goblet cell type on the one hand, or to a serous type of epithelium on the other; and this transition, whatever its cause, influences largely the nature of its secretion. Along with this fact I find in some cases a hyperplasia of the deep lying mucous glands—in other cases of the corresponding serous glands. The second point I intend to emphasize is that the inflammatory process in the various sinuses, and usually on the two sides, is of the nature forming in general, a connected picture of pansinusitis. In certain cases, of course, this process is limited to one or two sinuses, but more often is common to several

or all. Third: the main bulk of my demonstration will be devoted to the histologic changes in the tissue:

(a) Edema in all except the very chronic cases with extensive scarring;

(b) Epithelial hyperplasia, sometimes with goblet cell formation;

(c) Glandular hyperplasia, mucous or serous;

(d) Cellular infiltration, plasma cells being most common, then, in order of importance, lymphoid cells, eosinophile leucocytes, and neutrophiles. I am so far unable to corroborate the connection that some have established between eosinophilia and allergy;

(e) Changes in the periosteum and bone will receive little attention as I do not find them as important as some seem to find.

In a personal letter to Chapman, Davis brought out the fact that the lining membrane of all the sinuses is a delicate layer of periosteum immediately covering the bone, and upon which rests a thin, mucous membrane. Davis termed this lining of the sinuses mucoperiosteum.

According to Hajek, the inflammatory process in the paranasal sinuses in its extension to the bone has not been studied in all its details, and he thinks it is doubtful whether hyperplastic bone changes undergo resolution. He found that chronic inflammation of the lining of the sinuses caused periosteal irritation and formation of new bone, and all of these new bone formations he likened to the formation of osteophytes on the tibia in chronic ulcers of the leg. Hajek also thought that hyperplastic osteitis, even of mild degree, preceded rarefying osteitis. In discussing the extension of diseased processes from the bony framework of the sinuses, he said that the following possibilities must be considered:

(1) extension of the inflammatory process through the apparently completely intact bone; (2) extension by thrombosis of one of the large veins perforating the bone and spreading the infectious material to the other side of the bone; (3) through direct extension of the ulcerating process to the bone: periostitis, osteitis, and finally necrosis with perforation; (4) extension of the inflammatory process through dehiscences of the bony wall.

In the following reports a bit of history is given because it was thought that in some instances the history was significant; also, mention is made of the clinical findings as well as the gross and microscopic study of the pathologic tissue.

Much valuable aid has been rendered us by Dr. J. A. McIntosh, of the Pathologic Department of the University of Tennessee, in the preparation and study of the histologic specimens.

Case 1. Giles.—About four months prior to operation, the patient had an acute inflammation of all sinuses on the left. This attack promptly subsided under office treatment but the discharge continued as also did the headache. The patient had had discharge from the left side of the nose for several years but gave it no attention because he had no pain. After two months of treatment, a submucous resection and removal of the anterior end of the middle turbinate were carried out. The condition improved somewhat but the headache continued and did not improve.

Skiagram: All sinuses on the left side showed marked increase in density. Open operation on the left frontal, left ethmoid and sphenoid according to the method of Lynch was done February 28, 1927, under general anesthesia. The frontal extended to the superorbital notch and contained a much thickened mucosa and a good deal of thick yellow pus. The ethmoids were filled with a thickened granular mucous membrane and contained pus. The sphenoid was lined with a red, somewhat thickened mucosa. None of the sinuses were well developed.

Microscopic Examination: In the ethmoid specimen there was moderate hyperplasia of the mucosa and submucosa, hyperplasia of blood vessel walls, slight round cell infiltration of the stroma, and bony atrophy. The frontal specimens showed periosteal hyperplasia and bony hyperplasia.

Case 2. Gillette.—This patient was a young veteran of the World War and had had a chronic sinus disease of all sinuses since the influenza epidemic of 1918. He had had many sinus operations. A Killian type of frontal sinus operation had been done on the right side at some previous time and there was a history of several operations on the left frontal. There was some deformity on both sides but fortunately the frontal sinuses were small. The patient complained of so much pain on the right side that it was decided to reopen the right frontal and ethmoids. This was done January 7, 1927, an incision through the brow being used. A small frontal sinus was found filled with polypoid tissue and granulation masses. The anterior ethmoid cells had largely been removed. The posterior ethmoid cells were cleaned out at this time as well as the frontal and sphenoid. All of these sinuses were filled with a markedly thickened granular mucous membrane. At several points the mucous membrane was polypoid in char-

acter and all of the sinuses explored contained pus. Drainage was made into the nose by means of iodoform gauze.

Microscopic Examination: In the frontal specimen there was infiltration of the submucosa with eosinophilic cells, hyperplasia of the submucosa and the periosteum and bone. In the ethmoid there was found marked hyperplasia of the submucosa, infiltration with polymorphonuclear cells and small round cells and acute inflammatory exudate in scarred areas.

Case 3. Kennedy.—This young veteran of the World War gave a history of having had a discharge of pus from both sides of the nose since he had an attack of influenza in 1918. The skiagram showed all sinuses markedly increased in density. The frontals were large and extended almost to the line of the external orbital rim on both sides. An external operation was done on the right frontal, ethmoids and sphenoid December 7, 1926. All sinuses were found to be well developed and large, with thin bony walls. The frontal contained a much thickened mucoperiosteum. There was some polypoid degeneration of the mucosa above the frontal opening. The ethmoids were largely filled with polyps and some cells contained a thick granular mucous membrane and pus. The lining of the sphenoid was pink, thick and dull. There was no pus in the sphenoid.

Microscopic Examination: In the specimen taken from the frontal sinus the following was found: hyperplasia of the mucosa with infiltration by polymorphonuclear leucocytes and large numbers of infiltrating eosinophilic myelocytes. The ethmoid specimen was significant from the standpoint of much edema. There was also infiltration of plasma and eosinophilic cells, congestion of the blood vessels by polymorphonuclear and red cells together with hyperplasia of the blood vessel walls.

Case 4. Lerner.—A maiden lady, aged 50, gave a history of having had discharge from both sides of the nose since childhood. She had been treated for thirty years and had had intranasal ethmoid operations on both sides as well as bilateral intranasal antrotomy at some time in the past. She complained of unbearable headaches especially had with any acute attack of cold. External operation on the left side according to the method of Lynch was done in 1925. Bilateral Luc-Caldwell operation was done also in 1925. The right frontal was opened externally October 26, 1926, under general anesthesia. The frontal was moderately large and was almost completely filled with degenerated mucous membrane, polypoid in character. The superorbital ethmoid cells were completely filled with polyps. The sphenoid contained a thickened ocean water colored mucous membrane. Drainage was by iodoform gauze into the nose. The external wound was closed with dermal.

Microscopic Examination: The frontal and ethmoid specimens showed hyperplasia of the mucosa, submucosa and periosteum; lymphostasis and lymph channel dilatation; bony atrophy.

Case 5. Buckalow.—This patient, also a young veteran of the World War, gave a history of discharge of pus from the right side of the nose since having had an attack of influenza in 1918. He had headache with acute exacerbations of this trouble at more or less frequent intervals. The skiagram showed all sinuses on the right markedly increased in density and all sinuses large, well developed. External operation under local anesthesia, right side, was done December 16, 1926. The frontal sinus was found to be full of thick, yellow pus and the mucous membrane was thickened, red, and friable. The ethmoids were filled with large, dense polypi. The ethmoid cells extended far over to the orbit. The sphenoid was medium sized but unusually deep from front to back. The lining of the sphenoid was red and thick. Iodoform packing was used for drainage into the nose. The external wound was closed with metal clips. This patient expressed great relief from the time the operation was finished until he left the hospital two months later.

Microscopic Examination: In the ethmoid specimen the blood vessels of the stroma were several times thicker than normal. One area showed deeply stained, pinkish, widened basement membrane slightly infiltrated with polymorphonuclear cells. There were similar infiltrations into the surrounding stroma and the mucosa. And in some areas the mucosa was covered with necrotic pale pinkish debris. Some areas in the stroma showed elements of an acute exudate. The mucosa was slightly hyperplastic and the bones somewhat atrophic. The marrow spaces were increased in size and filled with edematous fibrous tissue containing many erythrocytes. The frontal specimen contained much the same as the ethmoid with more atrophy of the bone and much widening of the marrow spaces. These latter were filled with loose vacuolated edematous connective tissue. All of the soft tissue contained numerous fibroblasts.

Case 6. Dixon.—A graduate nurse, 24 years old, complained of headache on the left side and dizziness of increasing severity. She had had this trouble for more than four years. She had colds frequently and this made the headache and dizziness worse. Skiagram showed increased density in all sinuses on the left. Ethmoidectomy and submucous resection had been done about three years previously. External operation under general anesthesia was performed November 2, 1926. The frontal was small and easily cleaned out. It contained some mucus and a markedly thickened mucoperiosteum. The superorbital ethmoids contained thick granular membrane. The sphenoid was small but unusually long from front to back. All bone in this case was unusually thick.

Microscopic Examination: In the frontal specimen there were areas of epithelial metaplasia, atrophic mucosa, granulation tissue and acute inflammatory exudate of polymorphonuclear cells together with fibrin in the submucosa, dilated lymph channels, and one thrombosed vein. The ethmoid specimen showed hyperplasia of the mucosa, eosinophilic cell infiltration of the submucosa and periosteum.

Case 7. Engleberg.—A young married lady, 28 years of age, gave a history of severe headache and nasal obstruction for two years. She had had a bilateral ethmoid operation in 1925. The attacks of headache were growing worse and more frequent. External operation on the right frontal ethmoids and sphenoid was done under local anesthesia January 21, 1927. The frontal was medium sized and extended only slightly beyond the supraorbital notch. The lining membrane of the frontal was the thickest that I have ever seen. It was red, and roughly granular on the surface, almost filling the entire frontal sinus cavity. This lining membrane was removed in toto by submucous resection. The ethmoid cells were full of pus. There was no pus in the frontal except in a more or less isolated cell low between the two frontal sinuses. Closure of the wound was as usual and the recovery was uneventful. There has been no headache since the operation.

Microscopic Examination: The frontal specimen showed metaplasia and hyperplasia of the mucosa, connective tissue hyperplasia of the submucosa with infiltration of round and eosinophilic cells. There was considerable hyperplasia of the blood vessel walls and mucous gland atrophy.

Case 8. Littlejohn.—The patient had had a severe headache, principally on the left side, for a period of one week. The left eye had been swollen for three days. There had been dizziness and nausea, accompanied by profuse vomiting for three days. A left external ethmoidal operation was done under local anesthesia. There was moderate swelling of the soft tissue of the left orbit and lachrymal sac region. In elevating the periosteum the bone bled more freely than is usual. The frontal sinus cavity contained considerable pus; the mucous membrane was very edematous and was partially lifted off of its attachment. A large opening was made in the floor of the frontal sinus and the anterior ethmoid cells largely removed for good drainage. The mucous membrane of the ethmoids was very thick, partially filling the cells. Rubber tubing was placed externally in the wound and into the nose to insure drainage.

Microscopic Examination: The frontal and ethmoid specimens were much the same: edematous submucosa, infiltration with plasma cells and eosinophiles, and periosteal hyperplasia.

Case 9. Garner.—This patient was a young veteran of the World War who had been reported as having a large osteoma involving the frontal and ethmoid sinuses on the left. This bone tumor was ivory hard. We wish to present the pathologic report in brief as follows: The specimen consisted of numerous irregular bony masses and larger yellowish gray fibrous masses attached to some of the bony masses. On one surface, the numerous pieces of bone were smooth and polished, harder than compact bone. The fibrous masses were largely globoid in shape, the largest three centimeters in diameter and on section presented a smooth, shiny, moist surface attached to the periosteum of the bony fragments. One section showed a loose, edematous fibrous tissue covered by columnar epithelium, infiltrated by peri-

vascular round cells and eosinophilic cells. There were numerous small petechial hemorrhages scattered throughout. The histology was that of a benign, fibrous polyp showing congestion, edema and hemorrhage; chronic inflammatory fibrous hyperplasia with congestion, edema and multiple hemorrhages. There was much disorderly hyperplasia of the bone and extreme periosteal hyperplasia with adjacent granulation tissue.

Case 10. Bailey.—A young lady 18 years of age gave a history of having had arthritis in both arms and legs for nine years. During this time she had also severe attacks of headache. There was no definite history of sinus disease but the patient had considerable mucus discharge into the throat and continuous cold. At the first examination there was mucus on both sides of the nose, all turbinates were slightly hypertrophied, and the mucous membrane of the middle meatus was granular in appearance. The skiagram showed all sinuses more or less of the infantile type and increased in density. An external operation according to the method of Lynch was done under local anesthesia February 3, 1927. The mucous membrane of the frontal was pale and edematous. There was considerable mucus in the cavity. The mucous membrane of the ethmoids was thickened and the cells were filled with mucopus. The sphenoid was opened and the mucous membrane around the natural opening of the sphenoid was thick and swollen. Two weeks later the same operative procedure was carried out on the left side. The findings were practically the same. Later still radical operations were done on both antrums. The patient made an uneventful recovery having been much relieved.

Microscopic Examination: In the frontal and ethmoid specimens there was much infiltration of the mucosa and submucosa with plasma and polymorphonuclear cells, marked hyperplasia of the periosteum, bony atrophy and fibrosis of the marrow spaces. In the orbital wall there was noticeable bony hyperplasia.

Case 11. Quinn.—This patient was 64 years of age and had been troubled with purulent sinus disease for more than twenty-five years. A radical Killian operation was done on the right frontal eighteen years before this history was taken. For six months prior to this examination there had been much pain and swelling of the right frontal sinus and right eye. During this time there was an opening in the region of the right lachrymal sac through which yellow pus of foul odor had been draining. There was a marked depression of the right frontal sinus region and the skin over this area was red and swollen as was also the right eye. Both sides of the nose contained much pus and the mucous membrane was thick, edematous and to some extent polypoid in character. An X-ray showed marked increased density in all of the sinuses. In November, 1926, external operation was done under local anesthesia on the right frontal, ethmoids and sphenoid. The frontal sinus was partially obliterated but the mucous membrane present in the angles was red, granular and somewhat edematous in appearance. The cavity was filled with pus as also were the ethmoid

cells. There was a marked herniation of the nasointral wall which, when opened, was found to be filled with pus. Two weeks after the first operation the left side was operated on according to the method of Lynch. There was an opening in the bone of the floor of the frontal sinus and also of the ethmoids about the junction of the lachrymal with the ethmoid bone. The periosteum was rough and granular over these two areas. There was a hernia of the nasointral wall similar to the one described above. The lining membrane of the frontal and of the ethmoid was similar to that described for the right side. At a later date radical operations were done on both antra. Here also much pathology was found.

Microscopic Examination: In the right frontal the Haversian canals were increased in diameter and the marrow vessels were stuffed with polymorphonuclear cells. Some vessels were thrombosed and surrounded by numerous polymorphonuclear cells. In the left frontal there was bony atrophy, fibrous hyperplasia of the bone marrow and blood vessel walls. Some areas of hemorrhage were noted. In the right ethmoid there was epithelial metaplasia and hyperplasia. The submucosa was infiltrated and thickened by eosinophilic and plasma cells and fibroblasts. The left ethmoid showed atrophy of the mucous membrane, the mucous glands of the submucosa were dilated and filled with secretion. The lining epithelium was hypertrophied, finely granular and the surrounding stroma was infiltrated with plasma cells. The marrow spaces of the bone were widened.

Case 12. Cook.—This patient was a man 60 years of age who gave a history of having had for many years a discharge from both sides of the nose and discharge dropping back into the throat. About six months before our examination the patient had swelling of the face and eyes, worse on the right side. From this time he had severe headache and more discharge. At examination much scabbing was noted in both sides of the nose. The mucous membrane, after the pus and scabs were removed, appeared red and granular. The skiagraph showed marked increase in density of all sinuses, worse on the right. An external operation was performed under local anesthesia on the right side November 3, 1926. The mucous lining of the frontal was polypoid in character. There was considerable mucopus in the cavity. The lining of the ethmoid cells was thick and swollen but appeared to be less diseased than did the frontal. Drainage and closure of the incision was made according to our usual custom.

Microscopic Examination: The ethmoid and frontal specimens showed atrophy of the mucosa and plasma and eosinophilic cell infiltration and hemorrhage of the submucosa. There was also acute exudation of polymorphonuclear cells and fibrin in the bone marrow and thrombosis of the marrow blood vessels.

Case 13. Gills.—This patient, a man of 35, gave a history of having had much discharge from the nose with nasal obstruction for several years prior to examination. He also had had severe headache on the right side for three or four years. Vertigo was marked at times. Two

nasal operations without benefit had been done. At the time of examination there was much crusting in both sides of the nose but worse on the right. The middle turbinates were enlarged and there were polypi in the middle meatus of the right side. The skiagram showed marked increase in density of all sinuses on the right side. The increased density was especially marked in the frontal sinus. External operation on the right frontal, ethmoid and sphenoid was done under local anesthesia December 14, 1926. The mucous membrane of the frontal was thick, red and covered with mucopus. There was no partition between the right and left frontals. The ethmoid cells were filled with a thick, polypoid lining membrane. The mucoperiosteum lining the sphenoid was thick and dark red in appearance. Both the ethmoids and the sphenoid contained pus.

Microscopical Examination: The frontal specimen showed distinct ulceration of the mucosa, hyperplasia and hemorrhage of the submucosa. In the ethmoid specimen there were present hypertrophy of the mucosa, edema, and polymorphonuclear and round cell infiltration of the submucosa. There was an abscess in process of healing in the submucosa. A second ethmoid slide showed bony atrophy and hyperplasia of the blood vessel walls.

Case 14. Green.—This patient, a man 45 years old, gave a history of having had purulent discharge and obstruction of the left side of the nose for five years. At the time of examination there was much pus in the left side of the nose. The skiagram showed marked increased density in all the sinuses on the left. There was moderate increased density of all sinuses on the right. An external operation on the left frontal, ethmoids and sphenoid under local anesthesia was done January 7, 1927. The frontal was found to be large and practically filled with pus. The mucous membrane was dark red in color, very thick and polypoid around the frontal opening. It was difficult to remove the lining membrane from the upper angles. Small mirrors facilitated this step in the operation. The ethmoids were polypoid. The sphenoid was of moderate size. The mucous lining was thick and dark red in color. Drainage and closure was as usual.

Microscopic Examination: The frontal specimen showed marked hyperplasia of the mucosa and the submucosa, periosteal hyperplasia and bony atrophy. A specimen from the ethmoorbital wall showed atrophy of the mucosa and hyalinization of the basement membrane. There were dilated mucous glands lined with epithelium of a low cuboidal type, and marked plasma and eosinophilic cell infiltration of the submucosa.

Case 15. Ajax.—This patient, a widow of 50 years, had severe arthritis of various joints for fifteen years prior to examination. During this time she had been subject to colds and discharge from the nose and throat, as well as attacks of headache and pain in the cheeks and forehead. At examination pus was noted in both sides of the nose, especially coming from the frontal opening. The skiagram showed all sinuses increased in density, especially marked in the right antrum and

the right ethmoid. There were practically no frontal sinuses present. An external ethmosphenoidal operation was done under local anesthesia, on the right side, December 3, 1927. The mucous membrane of the ethmoids was thickened and these cavities contained mucopurulent secretion. Two weeks later a similar operation was performed on the left side, under local anesthesia. The mucous membrane of the ethmoids, sphenoid and the small frontal was quite thick and its surface was covered with tenacious mucus. Later, radical operations were done successively on the right and left antra, and when this was written the patient reported that her arthritis was more improved than it had been in eight years.

Microscopic examination of the left ethmofrontal wall showed extreme atrophy of the mucosa, condensation of the submucosa, bony atrophy, round cell infiltration, and hyperplasia of the blood vessel walls.

Case 16. Hall.—This patient was a man 35 years of age, who for eight or nine years had had crusting and purulent discharge from the right side of the nose. He had an ethmoid exenteration one year before this examination and the right antrum was irrigated several times four years previously. After the ethmoid operation the discharge of pus continued. Considerable pus was washed from the frontal sinus. He gave a history of vertigo, tight feeling of the head, and pain in the right frontal region. The skiagram showed increased density in all sinuses much more marked on the right side. A right ethmosphenofrontal operation was done under local anesthesia January 2, 1927. The mucoperiosteum of the frontal was thick, more or less edematous, and polypoid in appearance around the natural opening. There was thick, yellow pus present. The entire lining membrane was easily removed. The ethmoids and sphenoid had been satisfactorily cleaned up by previous intranasal operation. Drainage and closure was as usual, and the patient made a complete recovery from all symptoms.

The histologic examination of the frontal specimen showed goblet cell hyperplasia of the mucosa to be present, also subperiosteal hemorrhage and passive congestion of the bone marrow veins.

Case 17. McMurchy.—A married lady, 37 years of age, presented herself for examination October 3, 1926. She complained of having had pain in the left eye, left cheek and left temple together with watery discharge from the same side of the nose for three years. The discharge became purulent with each not infrequent attack of cold. For six weeks prior to examination she had "blind spells" accompanied by a sensation of pressure in the left eye. Examination revealed a badly deviated septum, tenderness over the left frontal, and pus in all parts of the left side of the nose. The X-ray showed marked increased density in all sinuses on the left side. A submucous resection and Halle intranasal frontal operation were done on the left side, followed in a short time by Luc-Caldwell antrum operation on the same side. There was no relief of symptoms and a left external operation was performed on the frontal ethmoids and sphenoid under general anesthesia February 10, 1927. The frontal sinus was large and ex-

tended to the hair line. The mucous membrane as a whole was not in an especially bad condition except at the outer angle where it was polypoid and covered with yellow pus. The superorbital ethmoid cells were extensive. On the posterior wall of the superorbital ethmoid cavity there was a cyst about the size of a hazelnut and was attached very firmly. The cyst contained bloody fluid. The sphenoid was very large and the lining membrane much thicker than normal.

Microscopic Examination: In the frontal specimen there was marked hyperplasia of the periosteum, bony atrophy, and marrow fibrosis. In the ethmoid specimen there was dilated cystic mucous glands, epithelial metaplasia, productive inflammation of the submucosa, and periosteal and bony hyperplasia. A specimen from the ethmoorbital wall contained marked blood vessel wall hyperplasia with obliteration of the lumen of some vessels, together with bony atrophy and bony hyperplasia. There was also fibrous hyperplasia of the bone marrow stroma.

Case 18. Rosine.—This patient, aged 32, had sinus trouble, worse on the left side, for the past thirteen years. She had had intranasal ethmoid and sphenoid operation on both sides without relief. She complained of severe pain about the right eye which was present practically all of the time, but there were acute attacks during which pain was more severe. There was discharge from both sides of the nose, worse on the right. At examination there was very little pathology seen in the nose. There was slight crusting about the right frontal opening. The X-ray showed moderate increase in density of all sinuses. An external operation on the right frontal, ethmoid and sphenoid was done under general anesthesia November 24, 1926. The frontal was small, the mucous membrane was thick and pale. There were several ethmoid cells far posteriorly which contained thickened mucosa and mucopus. Drainage and closure was as usual. Although the gross pathology appeared to be not very marked, this patient obtained complete relief from pain.

Microscopic Examination: The frontal specimen showed dilated marrow spaces in the bone and much perivascular infiltration of eosinophilic cells in the submucosa. In the ethmoid specimen there was atrophic mucosa, dilated lymph channels with edema of the submucosa, plasma and eosinophilic cell infiltration of the submucosa, atrophy of the mucous glands, and bony atrophy.

Case 19. Beard.—This patient was a 16-year-old boy, who stated that he had had a swelling of the frontal region in the median line practically all of his life. He had an intranasal operation about a year previously. For several years his breathing had been obstructed and there had been much purulent discharge from the nose. For several weeks there had been redness and swelling of the skin over the frontal and ethmoid regions on the right. At the time of examination the nose was obstructed by swelling and edema and discharge in both sides of the nose. There was redness and tenderness over the right frontal and ethmoid regions and a fullness over the

frontal region about one inch above the glabella. This enlargement was perhaps a little to the left of the median line. The X-ray showed marked increased density of all sinuses. Open operation was performed on the ethmoid and frontal of the right side under local anesthesia October 12, 1926. Pus was present beneath the periosteum. There was a rupture of the right frontal sinus in two distinct areas with considerable destruction of bone. An opening was made into the frontal for drainage and also through the anterior ethmoid cells into the nose. Rubber tubes were placed through and through for drainage. The patient rapidly improved and two weeks later the wound was reopened and the frontal sinus and ethmoids thoroughly cleaned out. The ethmoid cells and the lining membrane of the frontal sinus were polypoid in character. Iodoform packing was inserted for drainage and the external wound was closed. Two weeks after the second operation, the left frontal, ethmoids and sphenoid were opened under local anesthesia. The incision was made about one inch above the brow and carried over the swelling described above. A round cystlike tumor mass about one inch in diameter was removed. The cyst contained a small amount of fluid and was connected through a small opening in the bone with the frontal sinus. The usual incision through the brow was then made and the frontal sinus was found to be filled with a granulomatous mass. The bony wall between the cyst region and the frontal was entirely taken away. The ethmoids were large and extended entirely over the orbit. All the ethmoid cells were filled with polypi and swollen mucous membrane. The sphenoid was large, the mucous membrane was polypoid at the natural opening, and the mucosa was very thick. The wound was packed with iodoform gauze and the skin closed with dermal.

Microscopic Examination: In the frontal specimen were found typical granulation tissue, edema, and round cell infiltration. There were areas of connective tissue hyperplasia, some thrombosed blood vessels, and passive congestion of the marrow vessels.

Case 20. Gray.—A negro man, 35 years of age, gave a history of obstructed nose and discharge for four years or more, previous to January 11, 1927, at which time the first examination was made. For three months prior to examination he complained of headache almost constantly, and worse on the left side. There was much pus in the left side of the nose and the mucous membrane was red, swollen and granular. The Wassermann was positive. In the skiagram there was noted increased density in all sinuses, especially bad on the left side. The frontal sinuses were large. An external operation was performed on the left frontal, left ethmoids and sphenoid under local anesthesia February 25, 1927. The mucous membrane of the frontal was dark red and granular and thick. The whole lining membrane was removed in one piece. The ethmoids were filled with pus, thickened mucous membrane and polyps. The superorbital cells were very extensive, reaching over the orbit to the external limit of the frontal sinus. Closure was as usual and the recovery was rapid.

Microscopic Examination: The mucous surface was covered with pinkish granular debris mixed with red cells and fibrin. The submucosa was greatly thickened by connective tissue hyperplasia and contained numerous thick walled blood vessels. Some of these areas showed a predominance of connective tissue fibrils. There were several areas of small round cells and polymorphonuclear cells quite near blood vessels but there were no mucous glands present.

Case 21. George.—This patient was a graduate nurse 24 years of age, who complained of having had discharge from the nose, especially the right side, dropping of secretion into the throat and severe headache on the right side most of the time for one and one-half years or longer. For three months before operation there had been severe pain about the right eye and cheek accompanied by more or less frequent attacks of dizziness. The septum was badly deviated to the right and the mucous membrane of the whole right side of the nose was red and swollen. There was mucopus visible at every examination. In the skiagram there was noted increased density of the right ethmoid, sphenoid and especially the right frontal. There was a very dense area in the upper third of the right frontal sinus which was taken to be a beginning bone tumor. Operation was done through an incision in the brow, as several times detailed above, December 11, 1926, under local anesthesia. The mucous membrane of the frontal was thick and covered with mucopurulent material. The dense area spoken of in the X-ray was a glistening bony material being apparently an elevation and thickening of the posterior wall of the frontal sinus. The apex touched the anterior wall. The protrusion could be encircled with a bent probe. This protrusion or bony excrescence was studied by means of small mirrors and it was decided to leave it. The frontal sinus was completely cleaned of its lining membrane. The ethmoids and sphenoid linings were very thick, contained some granulation masses, and most of these cavities were filled or partially filled with pus.

Microscopic examination was not made as this interesting specimen was lost.

Case 22. Schadle.—This patient was a boy 7 years of age who was first seen eighteen months before operation, at which time there was a history of tears running from the right eye during his entire life. There had been purulent discharge from the eye most of this time. The lids and conjunctiva were red and somewhat thickened. Much pus could be expressed through the lower canaliculus by pressure over the lachrymal sac region. The lachrymal sac was removed shortly after this first examination. The wound healed promptly. After fourteen months the patient was again seen and the history obtained from the mother was that the eye had been discharging pus as before operation. At this time pus could easily be expressed. Under ether anesthesia April 14, 1927, an external incision was made over the lachrymal sac. There were granulations in the lachrymal fossa just posterior to which there was noted a large granuloma protruding through the bone. This mass was partially in the anterior ethmoid cells and extended through

an opening in the ethmoidal wall. The bone around this opening was necrotic and jagged. The granulated mass was removed. The ethmoid cells were entirely filled with thick granular mucous membrane. The posterior ethmoid cells were large, the lining membrane was thick and the cavities contained a moderate amount of mucus. There being no frontal sinus present, the ethmoid cells were thoroughly cleaned out, iodoform gauze was used for drainage into the nose, and the external wound was closed.

Microscopic Examination: A section of the ethmoid specimen showed folded mucosa lined with ciliated columnar epithelium and the submucosa contained numerous mucous glands. Some areas were infiltrated with polymorphonuclear cells while other areas were edematous and showed many fibroblasts, plasma and small round cells. The blood vessels were thicker than normal. The surface of the mucosa was covered by a granular pinkish debris with scattered areas of indefinite cellular elements. Other areas of the mucosa showed necrosis extending down into the submucosa. The adjacent muscularis showed fibrosis and hyaline degeneration of the blood vessel walls.

Case 23. Madison.—This patient was a young lady 18 years of age, who complained of having a cold all of the time. She was first seen April 4, 1927, and gave the history of having severe headache and difficult breathing for one year or more previously. The septum was deviated to the left and there was a large polyp filling the entire right nares and extending into the pharynx about one inch below the soft palate. The X-ray showed marked increased density in all sinuses on the right. An external operation as above described was done on the right side April 7, 1927, under local anesthesia. The frontal sinus was large, the lining membrane edematous, and the cavity contained pus. All of the ethmoid cells were filled with edematous mucosa and polyps. The superior ethmoids were extensive. The vessels coming through the anterior ethmoidal foramen with the nasal nerve were ligated with a new needle. The sphenoid was lined with edematous mucous membrane which was red.

Microscopic Examination: In a specimen taken from the mucous membrane of the frontal floor there were some areas of superficial desquamation leaving only a single layer of epithelium. There were areas also composed of stratified, low cuboidal epithelium. Other areas showed thick, hyalinized basement membrane with the adjacent submucosa containing numerous thickwalled blood vessels, fibroblasts and infiltrating round cells. In still other areas there were streaks of hyalinized connective tissue. There were also numerous plasma and eosinophilic cells, polymorphonuclear infiltration, and the adjacent bone was covered by a thickened hyperplastic periosteum. The second specimen was from the lining membrane of the sphenoid. This section showed areas of ulcerating and dense purplish-black areas of lime salts deposit. These deposits were scattered throughout the submucosa. There were areas showing marked connective tissue hyperplasia and hyaline degeneration. The blood vessels were many times thicker than

normal. The characterizing features were productive inflammation, hyaline degeneration, and lime salts deposits.

SUMMARY.

In the twenty-three cases reported there were found two cysts, one osteoma, no cholesteatoma; twelve were polypoid, there was bony necrosis to the point of perforation in three, periostitis in twenty, one granuloma, no mucous membrane tumor, and one case had no partition between the two frontal sinuses. Polypoid degeneration of the lining of the frontal sinus may take place first and principally at the outer angle in large frontal sinuses, and also about and above the natural opening in any sized cavity. The use of pharyngeal mirrors has been of much service to us in the removal of small tags of soft tissue from the frontal sinus; especially was this true in high, narrow cavities where direct vision was impossible. In a general way the diagnosis in these cases was confirmed by the operative and histologic findings. There was ulceration of the mucous surface in several cases as was demonstrated by the microphotographs. Metaplasia of the mucous membrane was present in a good percentage of the twenty-three cases reported. Goblet cell hyperplasia was particularly marked in one case and this was associated with subperiosteal hemorrhage. Prolonged chronic irritation of the mucous membrane was apparently responsible for metaplasia changes. Hyaline degeneration of the basement membrane was present in several cases. In the submucosa in nearly every case there were present many thrombotic blood vessels, blood vessels with hyperplastic walls, some hyaline degeneration of the blood vessel walls, dilated lymph channels, and atrophic and dilated mucous glands. The submucosa was hyperplastic in all cases and contained polymorphonuclear cells, plasma cells, fibroblasts, small round cells, eosinophiles and eosinophilic myelocytes varying in the several cases. The periosteum was markedly thickened in every case. In some cases it was ten or fifteen times thicker than normal. Subperiosteal hemorrhage was present in at least one case. In the bone, resorption with widening of the Haversian canals and marrow spaces, together with roughening of the edges of the bony walls, were common findings. There was lymph channel

dilatation in the bone marrow in at least two cases. Blood vessel thrombosis with obliteration of the vessel lumen in the marrow spaces and also in the submucosa was present in several cases of acute osteomyelitis. In one instance there was obliteration of a vessel and partial organization of the clot suggesting one method of total occlusion of the blood vessels. There was hyperplasia of the bone in several cases and exostosis demonstrable in at least one case.

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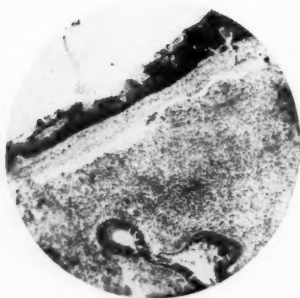


Fig. 1. Ethmoid. Superficial ulceration of the mucosa. Hyperplasia of submucosa. Periosteal hyperplasia and bony resorption or atrophy.

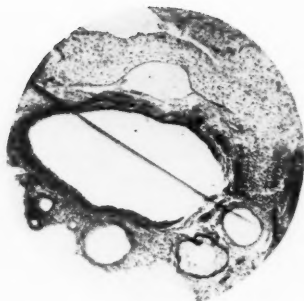


Fig. 2. Ethmoid. Cystic dilatation of mucus glands and ducts.

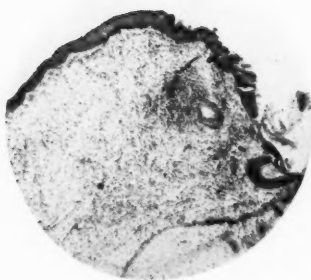


Fig. 3. Ethmoid. Epithelial metaplasia and hyperplasia. Chronic inflammation of submucosa.

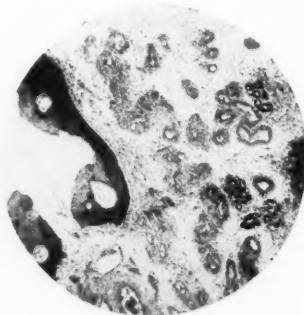


Fig. 4. Ethmoid. Atrophy of mucus glands. Vascular wall hyperplasia. Periosteal hyperplasia. Bony resorption or atrophy.

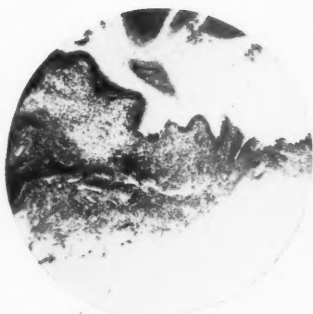


Fig. 5. Ethmoid. Atrophy and metaplasia of mucosa. Fibrosis of submucosa with round cell infiltration. Vascular wall hyperplasia.

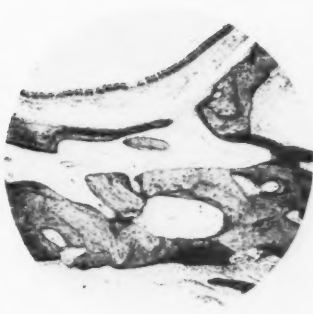


Fig. 6. Frontal atrophy of mucosa. Bony hyperplasia. Lymph channel dilatation.

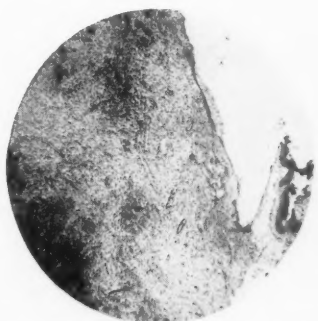


Fig. 7. Frontal sinus. Chronic productive inflammation with submucosal hemorrhages.

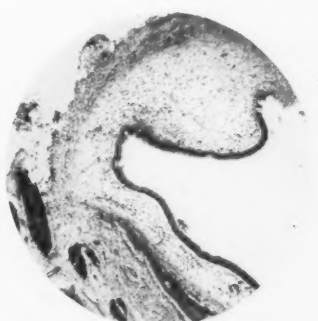


Fig. 8. Frontal sinus. Plasma cell infiltration and edema of submucosa. Periosteal hyperplasia and bony resorption.



Fig. 9. Frontal. Acute osteomyelitis. The darkest area is a thrombosed vein. In the lightest area the exudative elements have degenerated.

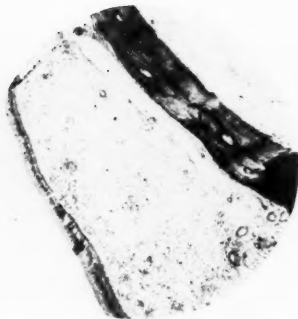


Fig. 10. Frontal. Acute peri-vascular exudation in the submucosa.

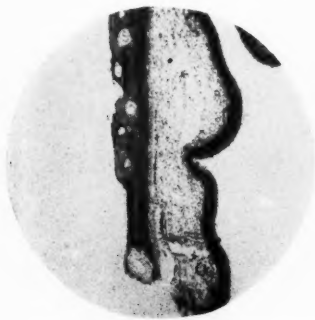


Fig. 11. Ethmoid. Edematous submucosa. Hyperplasia of the periosteum and widening of the Haversian canals (atrophy or resorption).

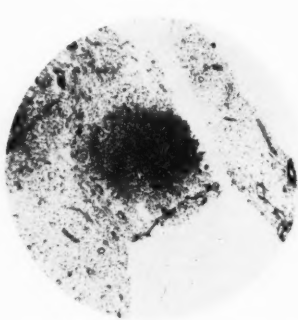


Fig. 12. Frontal. Healing abscess in the submucosa. Blood vessel wall hyperplasia. The circumscribed cellular area has thin walled capillaries. Fibroblasts and polymorphonuclear cells.



Fig. 13. Frontal. Superficial ulcer of the mucosa and chronic inflammation of the submucosa.



Fig. 14. Ethmo-orbital wall. Marked periosteal hyperplasia. Bony hyperplasia and wide marrow spaces filled with hyperplastic connective tissues.

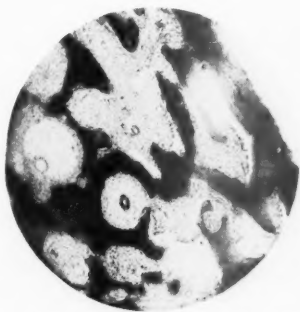


Fig. 15. Ethmo-orbital wall. Bony hyperplasia and widened marrow spaces.



Fig. 16. Ethmo-orbital wall. Ulceration of the mucosa with hyaline degeneration of the basement membrane, especially marked where a single layer of low epithelium exists. Plasma and polymorphonuclear cell infiltration.

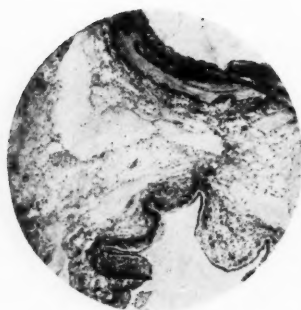


Fig. 17. Ethmo-orbital wall. Ulceration of the mucosa with marked hyaline degeneration of the basement membrane. Edema of the submucosa.

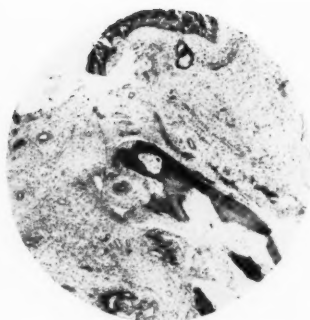


Fig. 18a. Frontal. Bony atrophy or resorption. One large giant cell osteoclast in a dilated Haversian canal and a nearby blood vessel with greatly thickened walls. Round cell infiltration of the submucosa.

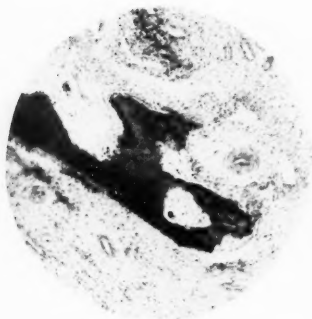


Fig. 18b. Frontal. High power showing the osteoclast in the dilated Haversian canal.

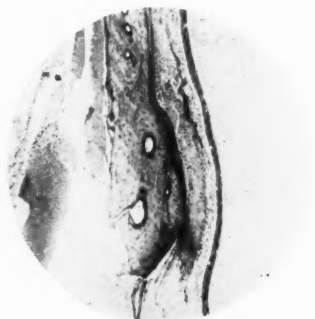


Fig. 19a. Frontal. Subperiosteal hemorrhage adjacent to a bony exostosis suggesting that the latter may have developed subsequent to hemorrhage which became organized into bone.

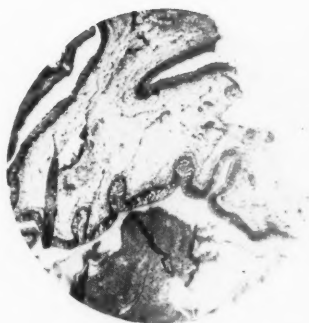


Fig. 19b. Ethmoidorbital wall. The mucosa with goblet cell hyperplasia. Vascular wall hyperplasia in the submucosa.

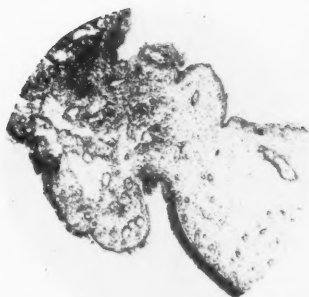


Fig. 21. Ethmoidorbital wall. Superficial ulceration of the mucosa. Edema of the submucosa. Plasma and eosinophilic cell infiltration of the submucosa.



Fig. 22. Frontal. Thickened blood vessels and bony atrophy. Moderate edema of the submucosa.

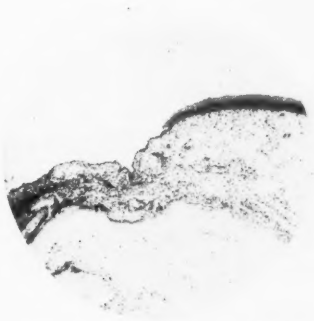


Fig. 23. Frontal sinus. Ulcer of the mucosa. Edema of the submucosa with plasma and eosinophilic cell infiltration.

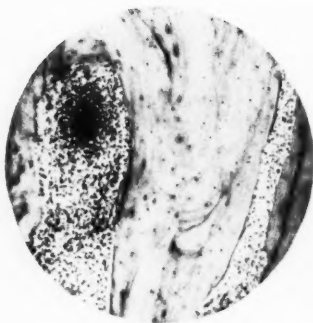


Fig. 24. Frontal. Acute osteomyelitis. The dark area is a thrombosed blood vessel.



Fig. 25. Frontal. Excessive disordered bony hyperplasia. Clinically so called osteoma.

XXXVII.

INFECTION OF THE SPHENOID SINUS.*

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The sphenoid sinus as a center of infection has never been given sufficient emphasis, either by the medical profession or the laity, but the average patient understands about, in fact, can converse quite glibly on, frontal sinusitis and the antrum. Most physicians on encountering frontal or maxillary sinusitis are quite anxious to call in consultation the otolaryngologist, and I feel justified in stating that in many cases the sphenoid sinus is entirely neglected; at least, its significance is minimized. I confess that I have not always realized its rôle or been cognizant of it.

The literature is not voluminous on sphenoid sinusitis, and some authors dwell principally on the roentgenographic diagnosis. Lobell has written illuminatingly on the puncture irrigation diagnosis of sphenoiditis, and Shea has made a comparison of the various methods of surgical procedure.

The sphenoid sinus develops in the body of the sphenoid bone posterior to the ethmoid labyrinth from about the eighth to the tenth year of life; it assumes the same relationship to the posterior ethmoid sinus as does the maxillary to the anterior ethmoid; that is, it not infrequently acts as a reservoir for the suppurative drainage from the posterior ethmoid sinus. Before adolescence the sphenoid sinus develops rapidly, and in adult life may be equal in size to the maxillary sinus.

The sphenoid sinus is first of all rather inaccessible, and because of its propinquity to vital structure it is not wise to approach it without adequate knowledge of the anatomy. The

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posterior wall of the sphenoid sinus is covered first with meninges; then the cavernous sinus and its contents, the carotid artery, and adjacent to that the pituitary gland and optic nerve are in intimate contact with its wall. In view of the fact that all these important structures are practically contiguous to the sphenoid sinus it should cause less wonder why ocular involvement and meningitis occur as sequelæ than that such consequences do not occur more frequently than they do!

Schaeffer, Loeb and Lobell have found that most sinuses are of average size or larger; the small sphenoid sinus is more rare. The smaller the sphenoid sinus the more nearly it is situated on a level with the ostia and drainage is good; therefore the small sphenoid sinus only infrequently comes under the observation of the rhinologist.

On examining wet specimens, skulls and roentgenograms, one notices that in many instances the size and capacity of the sphenoid sinus approach the size and capacity of the maxillary. The combined volume capacity of the sphenoid sinuses and antrums is greater than that of the ethmoid and frontal sinuses.

The symptoms of sphenoid infection are quite distinct and characteristic. As in all sinus infections, there is the feeling of fullness and heaviness in the head, especially on stooping. The voice lacks normal resonance. The pain and ache are most pronounced under the occiput and in the temporal region; the patients frequently complain of an ache in the frontal region, about midway between the level of the base of the nose and the hairline. Walking, or jarring of the head, is accompanied by marked pain and discomfort. There is also a so-called Vidian syndrome not unlike Sluder's sphenopalatine picture. The pain extends into the ear and mastoid, and in some cases to the scapula and arm. This Vidian syndrome is more frequently encountered in the chronic and hyperplastic type.

It may occur frequently that the rhinologist has washed the antrum and cleaned the anterior group of sinuses and still the patient's discomfort continues until there is a spontaneous evacuation of pus; the patient states that "something gave way" in his head and he is relieved. In most instances this is spontaneous emptying of a suppurating sphenoid sinus.

When a normal sphenoid sinus is opened there is seen a grayish mucous membrane lining. If the sinus is infected the membrane becomes markedly reddened. According to Warlow, who has made extensive microscopic studies of sphenoid specimens, a small amount of pus or a small localized area of infection may cause marked symptoms, so that it is not necessary to encounter macroscopic pus during the investigation. The exudate is not pouring from the sinus in a stream but collects, empties and gradually fills again. Thus, the physician is fortunate if even on repeated examination he sees the field just at the time when pus or exudate is being evacuated from the sphenoid sinus. It is not safe to pronounce a sphenoid sinus as normal on just one examination, especially if there is any suspicion at all.

Examination of the nose may reveal only congestion. The middle turbinate may occlude the view; if so, there is no contraindication to crowding it laterally. The anterior surface of the sphenoid sinus is sensitive to palpation and frequently as the examining probe causes pressure over the sphenoid sinus the patient will state that the pain in the occiput or temporal region is excited. Some patients complain of pain in the shoulder blade when there is undue pressure on the sphenoid. One occasionally sees a small quantity of pus between the middle turbinate and the septum, too high to have come from the middle meatus; more commonly, however, the patient complains of the pus dropping into the throat from the pharynx, and pharyngeal examination may reveal an accumulation of pus over the anterior surface of the sphenoid sinus.

Roentgenograms are helpful in visualizing and outlining the size and extent of the sphenoid sinus; furthermore, and this is probably more important, they show the variation in size and configuration of the sinuses on the two sides. No two are exactly alike: The right sphenoid may occupy almost the entire sphenoid body, while the left occupies just a small area; one cavity may have a partition or the septum may be in a diagonal position. It seems, therefore, most necessary that the roentgenogram be carefully studied in each case, especially if any surgical procedure is to be carried out or diagnostic puncture and irrigation are to be instituted. (Figs. 1 to 4.)

The technic, as carried out by Dr. Sisk, who has cooperated with me in this work, has proved satisfactory. A lateral view is obtained to show the extent and outline of the sinuses in the anteroposterior direction, and a roentgenogram is taken in the vertical position with the plate under the chin and the tube above the head, directing the rays in such a way that the shadows of the sphenoid sinuses are projected within the arch of the jaw. In this position the size and the extent laterally are revealed satisfactorily; also the position of the septum between the two sinuses is demonstrated.

I am very much in favor of, and use, the diagnostic puncture method of Lobell in suitable cases. The sphenoid body is cocaineized, the head held in the erect position, and Lobell's cannula introduced on a line from the anterior nasal spine past the posterior end of the middle turbinate and hugging the septum. One need have little apprehension, as the guard on the cannula will stop the progress of the needle well within 7 mm. of the punching point. Gentle air pressure or sterile solution of sodium chlorid may be used to evacuate the contents of the sinus. If microscopic examination of the purulent contents is desired, the material may be obtained. It is not necessary to remove the middle turbinate; if it is obstructing it may be crowded to the lateral wall, thus allowing adequate approach to the sphenoid body.

Recurrent attacks or chronic sphenoid sinusitis, with or without hyperplastic changes, present a problem difficult to solve. The symptoms as they have been described still exist but are not quite so intense. In addition, there is a dry pharynx with occasionally atrophic changes, and the development of secondary laryngitis with hoarseness is not unusual. There may or may not be ocular symptoms. Men of unquestioned authority have reported excellent results in clearing disorders of the eye by operating on the sphenoid sinus, while others of equal rank maintain that they do not believe that definite changes occur in the eye but that there may be neuralgic symptoms. It is not in the province of this paper to discuss the controversy. However, it seems logical to suppose that a sinus which is so intimately associated with the course of the optic nerve might in the presence of infection in some way cause symptoms of disturbance of vision.

One of the most annoying features is crusting in the pharynx. The patient finds this difficult to dislodge; gargling is ineffectual. The only relief he gets is from nasal irrigation. Of course, this mechanically washes the crust free but has no effect on the sinus infection.

If the lining of the sphenoid sinus becomes polypoid there is considerable discomfort and pain of the Vidian type.

The treatment for inflammation of the sphenoid sinus in the acute stage is the same as in other sinuses. This includes such procedures as shrinkage, suction and steam inhalation. The method I am using at present is to wash out the sinus with the cannula as was just described. If this gives only temporary relief and there is a tendency for recurrence, it is necessary to enlarge the natural opening with one of the approved sphenoid punches. This opening is made temporally and downward. Usually the symptoms subside and the pathologic condition improves as soon as an adequate opening allows free ventilation. Frequently the opening remains patent for many months or even years.

In the cases of chronic sphenoid sinusitis the treatment must be instituted in a more extensive and radical manner. Inflammation of the sphenoid sinus is less commonly found alone than in association with a pathologic condition in the postethmoid group. There are two types, suppurative and hyperplastic. In the suppurative type there are symptoms of postnasal discharge, dry pharynx and some coughing. Headache does not seem to be a constant symptom unless drainage is obstructed.

In the hyperplastic type there are more pronounced symptoms. It is this type of case which gives the well known Vidian syndrome of pain in the neck, shoulder and scapula. Occasionally there is some pain in the eye but this is not constant. Otalgia is common; Lyman reported cases of otalgia or mastoidalgia in which relief followed on opening the sphenoid sinus.

Surgical attack should be quite complete; the ethmoid sinuses are opened and the procedure carries the operator through the entire capsule to the ethmosphenoid septum. It may or may not be necessary to remove the posterior third of the middle turbinate; rarely is it necessary to remove the entire turbinate. The operation should carry through into the sphenoid sinus.

noid sinus, and the anterior wall of the sphenoid cell should be opened wide. Care should be exercised if the lining of the sinus is disturbed, as often an artery of large caliber is encountered, and if this is damaged the bleeding would be difficult to control. As a rule, adequate ventilation seems to accomplish as much in itself as the method of using various astringent drugs and chemicals. In fact, a 1 per cent solution of phenol in mineral oil has a soothing and beneficial effect.

The after care is fraught with many problems. There is a tendency on the part of the hyperplastic tissue to recur in small buds, sometimes closing the sphenoid sinus or filling in from the back of the sphenoid. This soft tissue springs up within a few weeks. If hyperplasia should persist the application of radium, 100 milligram hours' exposure, changes the myxomatous tissue to fibrous tissue and tends to discourage the growth of the polypoid structure.

Patients operated on in the manner just described are quite comfortable, but find it necessary to visit the rhinologist at intervals for relief, as even under most favorable circumstances granulation and hyperplastic tissue tend to regenerate.

CONCLUSIONS.

1. Infection of the sphenoid sinus is more common than it has been wont to be regarded.
2. No study is complete without careful survey of the roentgenograms.
3. Diagnosis may include puncture and washing.
4. Surgery, well directed, is no more hazardous than in the case of maxillary sinusitis, and in chronic cases should be quite radical.

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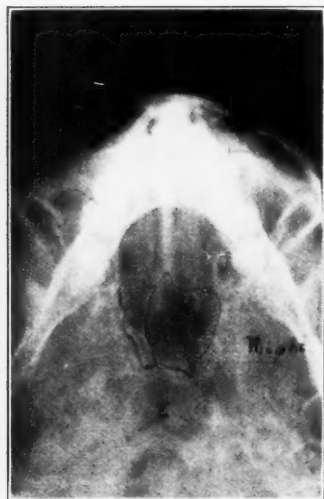


Fig. 1.

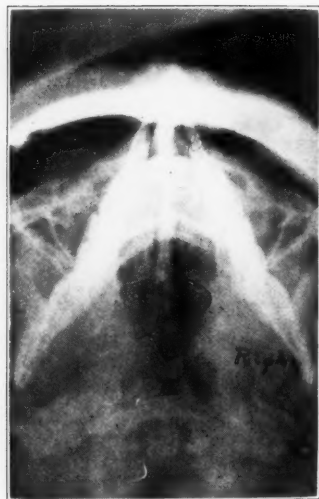


Fig. 2.

Figs. 1 and 2. Roentgenograms from vertical position showing outline of sphenoid sinuses. Outline is traced to accentuate borders.



Fig. 3. Roentgenogram of skull from vertical position. Right sphenoid sinus is filled with lipiodol. Left sphenoid sinus is much larger than the right. Outline is traced to accentuate border.

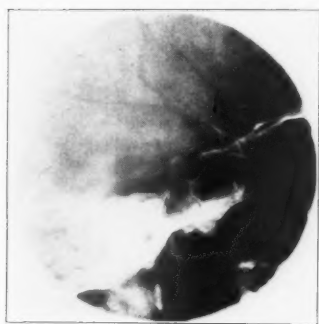


Fig. 4. Lateral view of sphenoid sinuses, retraced to accentuate outline and to demonstrate overlapping.

XXXVIII.

FRACTURES OF THE FACIAL BONES INVOLVING
THE NASAL ACCESSORY SINUSES.

By J. B. NAFTZGER, M. D.,

SIoux CITY.

Our thought in presenting this subject is not that we are introducing something entirely new, but rather to bring before the rhinologist a condition little discussed in our literature and which occurs with sufficient frequency to engage our consideration. In reviewing the literature of the last few years, we found that the dental and the oral surgeons have contributed practically everything written along this line, although during the war numerous articles were written by military surgeons and dental and oral surgeons.¹ Some of the textbooks on general surgery have given some attention to fractures of the face.² While the oral, general and dental surgeons have taken care of the facial fractures, it seems, inasmuch as the structures involved are distinctly in our field of work, and the nasal accessory sinuses are as a rule involved, we should study the treatment of these cases and at least assist in the restoration of form and function of these parts. When the walls of the sinuses are comminuted and the sinus filled with blood, an infection soon develops so that a skilled rhinologist should supervise the treatment and after care. Especially in the smaller centers, where an oral surgeon is not available, the rhinologist sees many cases of fractures of the facial bones. In our experience the majority of these fractures of the facial bones are due to automobile accidents, and with the increase in cars and speed, accidents are becoming much more frequent. A certain percentage is due to industrial accidents, athletics and sports. The incident of auto injuries has greatly increased in the last few years. In a paper published in October, 1918, I reported a number of cases of fracture of the face involving the antrum and only one of these was due to an automobile accident.³

In every community can be seen cases of unsightly deformities following injuries which involved the face. From both a social and a business standpoint these deformities are a great handicap to the unfortunate victim. Manifestly something has been neglected in the treatment of these cases, and we feel that the rhinologist should play a more important part in this work and should be prepared to take care of these mutilating fractures and the sinus complications which usually follow.

As these injuries are usually accompanied by other body injuries a general surgeon has charge of the case, and quite frequently the nose and face fractures are not reduced because the surgeon's attention is centered on the more serious complications. As a result, should the patients survive, they are more distressed by the appearance of a deformed face or nose than by the more serious but less apparent injuries, and later begin to look for relief in plastic surgery. Facial injuries are usually accompanied by swelling and edema, and when this subsides the external deformity and disturbed function become more apparent. Fractures of the nasal and the facial bones unite quite early, so that the displaced bones should be reduced early. By the second week it is hard to reduce the fractures and keep them in place, while as a rule they are easily reduced if taken early.

This discussion will be limited to crushing injuries involving the bones of the face, with or without laceration of the soft tissues and without extensive loss of the soft structures. The bones most frequently involved are the nasal, superior maxilla and the malar. The maxillary antrum and the ethmoid are very frequently involved and occasionally the frontal sinuses are injured. Thus the surgeon is confronted by the problem of treating the sinuses which frequently become infected as well as reducing the fractures of the bones.

PATHOLOGY.

In the fractures of the superior maxilla the alveolar process may have a fracture involving a part of the alveolus or a horizontal fracture may separate the entire alveolus and palatine process from the maxilla with the exposure of the antral and the nasal cavities; and there may also be a separation of the palatine processes of the maxilla. There may be a transverse

fracture through the malar bones, orbit and the nasal bones in which the entire upper jaw and face is movable. In these two types of fracture the teeth may follow the excursion of the lower jaw, dropping down from one to two cms. when the mouth is opened.

Three such injuries have come under our observation in the last year, all auto victims. In two, the entire superior maxilla was detached from the skull by the transverse fracture, and the upper face was movable, and, due to the displacement downward, the face appeared much elongated, giving a very unnatural and ghastly appearance. In the other case there was a complete separation of the alveolus and the palatine process from the maxilla, allowing the teeth to drop downward when the mouth was open and exposing the antral and the nasal cavities to view through the lacerated mucosa under the lip.

The maxillary sinuses are frequently involved by either vertical fractures or more commonly by comminution of the outer wall, the malar bone being frequently driven medially into the antrum. Several of these cases have come in following football injuries and a few from kicks from horses, and in one the victim fell, striking the malar prominence on a cement floor. While the maxillary sinuses are most frequently involved, the ethmoid and frontals may also be fractured. A fracture may extend through the ethmoid labyrinth, or the ethmoid cells may be crushed. The frontal sinus may be opened by separation of the floor or may have a fracture extending through the anterior wall or the posterior wall.

The malar bones may be fractured or driven medially into the antrum. The displacement follows the direction of the injury. Only rarely are both malar bones fractured, in our experience, although in two cases both were fractured. There is usually an irregularity or displacement of the infraorbital ridge, and there may be a displacement of the lateral orbital wall inward. In two of the cases with the transverse fracture with separation from the skull, the orbital floor being displaced downward, the eyeball was also displaced downward and outward. As a rule, the solid malar bone is driven medially into the antrum; the anterior wall of the antrum and infraorbital ridge are crushed and buckled. The fracture may extend into the nasal cavity and ethmoid as well as the orbit. In some of

these fractures of the malar there seems to be a displacement or fracture at the articulation of the frontal as well as the zygoma, but there may be a fracture at other points. The bone is usually driven in the direction of the applied force, so that in rare cases it may be driven into the orbit.

The nasal bones in severe injuries may be completely crushed, causing marked depression with disarticulation from the frontal bone above, and in these severe injuries the ethmoid is usually fractured. One nasal bone may be disarticulated from the maxilla laterally or from the opposite malar bone medially, causing a lateral displacement. Fractures of the nasal bones are usually comminuted and often compound. The vertical plate of the ethmoid and vomer and the cartilages of the nose are often fractured, the ethmoid labyrinth may be opened and the fracture may extend back into the sphenoid or to the base of the skull. In any of these fractures we may have edema and emphysema, which may involve the orbit, and a subcutaneous or subconjunctival hemorrhage. The eyelids, eyeball and the orbital contents may be injured in connection with the fractures of the bones.

Through the courtesy and assistance of Dr. G. R. Albertson, professor of anatomy at the University of South Dakota, we attempted to produce fractures of the facial bones on the cadaver to determine, if possible, the type and extent of these fractures. While manifestly the nature of the injuries was not identical, we attempted to apply the force in the directions indicated by the study of the cases that have come under our observation. A short description of the extent of these injuries in a few cases may be interesting.

First experiment: The force was applied directly on the lateral surface of the malar with the following findings: Malar disarticulated from the frontal and zygoma; vertical fracture of facial surface of maxilla; orbital floor fractured; orbital process of malar driven into the maxillary sinus and the coronoid process of the lower jaw was fractured.

Second experiment: The malar bone was driven downward and backward from a blow on the infraorbital margin. Findings: The frontal process of the malar was broken below the articulation with the frontal; facial surface of maxilla fractured vertically; zygoma fractured at its junction with the squa-

mous portion of the temporal bone, and there was a lateral displacement of the zygomatic arch.

Third experiment: Force applied over the bridge of the nose; nasal bone crushed with crushing or buckling of the upper part of the nasal septum (vertical plate of ethmoid) with a fracture of the cribriform plate of ethmoid and loosening of the crista galli; separation of frontal processes of the maxilla from the nasal process of the frontal, thus opening the floor of the frontal sinus; the maxillary sinus may or may not be opened; the ethmoid labyrinth was fractured and comminuted. The pterygoid process was broken from the body of the sphenoid, thus opening the sphenoid sinus. The malar was disarticulated from the frontal and the palate was fractured.

Fourth experiment: Force applied to the alveolar process from the front; both maxilla were fractured horizontally through their bodies, exposing the maxillary sinuses; the pterygoid process was fractured below and not from the body of the sphenoid, and therefore the sphenoid sinus would not be opened unless it extended down into the process; the nasal septum was fractured from before backward through the septal cartilage and vomer.

Other experiments were made, but these described were the most typical of the injuries coming under our observation. It was demonstrated, first, that the sinuses were involved in almost every fracture of the face; second, that fractures of the cribriform plate can occur from a crushing fracture of the nose when the force reaches the vertical plate of the ethmoid; third, the sphenoid sinus was apparently opened by a separation of pterygoid process from the body of the sphenoid; fourth, when the malar bone is driven medially the orbital plate and not the body of the malar bone is driven into the maxillary sinus.

We fully realize that these experiments are incomplete and not of sufficient number to justify making statements or forming conclusions so that we are simply describing the conditions produced.

DIAGNOSIS

In our first examination of these fractures of the face a careful inspection is important. This inspection must include the

contour of the face, the alignment of the teeth, the infraorbital ridge, the malar bones and the zygomatic processes. If the teeth drop downward on opening the mouth we must determine whether there is a transverse fracture with separation from the skull or a fracture involving the alveolus and the palatine processes only. There are usually multiple lacerations of the soft tissue and openings may extend through the comminuted bones in the antrum, ethmoid or nasal cavities and in some instances into the frontal sinuses. There is usually considerably edema which changes the contour over the normal bone and obscures the fracture, thus adding difficulty to the diagnosis. By digital palpation crepitus can usually be elicited in these fractures. If the case is not seen until several days following the injury and there is continuous pain and considerable swelling over the antrum and side of the face it usually indicates a fracture in our experience. A radiogram is of value if properly taken but unfortunately in many of these cases it is impossible to get a satisfactory radiogram due to the extensive injury and the condition of the patient.

TREATMENT

Lacerations, if extensive, must be sutured, but sutured loosely to provide drainage, particularly if the tissues are much mutilated. Where the edge of the laceration is very ragged it is better to trim off the loose tissue before suturing. The facial tissues have apparently a greater immunity to infection than do other tissues, due to the abundant blood supply, so that the end results are more gratifying than would at first seem possible. It may be necessary to put in a small drain such as silkworm if the laceration is extensive and extends into the nasal cavity or sinuses. The eyelids demand especially careful approximation.

The bony fractures are the most important and demand immediate attention. Early reduction and immobilization of the fractured parts reduces shock, relieves pain, controls hemorrhage and gives a more rapid restoration of function. We shall not attempt to describe or illustrate all of the appliances which are advocated in the treatment of these fractures. In the first place a dental surgeon is indispensable if the alveolar processes

and teeth are involved, or if there is a transverse fracture of the superior maxilla with separation from the skull. The dental or oral surgeon is much more familiar with the making of the splints and appliances necessary to reduce and hold these fractures. The fractures as we have mentioned are as a rule multiple, often comminuted and frequently compound. Where the alveolus only is fractured the dental or oral surgeon can apply interdental wiring where the patient has the teeth in place and in edentulous cases the plate can be used if it is not lost. This plate can be used with a dental tray and molding compound and this is attached to a suitable headgear. This headgear may be made of various materials as surgical web, tape or adhesive plaster. The rigid headgear made of guttapercha and tape as advocated by Dr. V. H. Kazanjian⁴ is probably the most satisfactory. In this a guttapercha band is molded to the forehead and secured by surgical tape around the head. To this guttapercha band attachments can be made for elevating the depressed bone or exerting pressure where needed. I will later show modifications of such appliances. In some cases I have shaved the head and placed a broad adhesive tape from the back of the neck to the forehead and over this one or two layers of adhesive type is placed, giving opportunity for attachment of the supports. Silkworm suture or wire can be attached to a tooth, brought out through the cheek and attached to a headgear. This, however, allows but one angle of traction and could not be adapted in all cases. In complete fractures, where the maxilla drops downward or the alveolus is separated, a Kingsley splint can be placed in the mouth and wired to the teeth, if present, and the arms of his splint, which extend outward on either side of the mouth, are attached by tape and rubber bands to the headgear. In cases not too extensive it is often possible to hold the jaw in place by using a chin piece which is attached to a headgear, thus using the lower jaw as a splint. The objection to this method is that it is hard to overcome the muscle action of the lower jaw, feeding is unsatisfactory and breathing is usually difficult, as the nose, as a rule, has been injured and is occluded by packs and swelling. Where the external wall of the antrum is comminuted and depressed and the malar bone driven into the antrum, we open above the alveolus, as in a radical antrum operation,

and elevate the fractured bone. When the malar bone has been driven into the antrum, grasping it with a hooked tenaculum externally can be combined with this method. All pieces of bone that have any firm attachment are pushed up into place, but the pieces which are loosened almost entirely are removed. As a matter of fact, considerable bone can be removed without any noticeable after effects. Because of the abundant blood supply in the face, necrosis of the bony fragments rarely occurs. After replacing the bone, vaselin gauze is placed in the antrum through the opening made above the alveolus. This can be left in place several days before removing and repacking, and we have found that at the time of the first dressing there is little danger of the bony fragments becoming displaced, as already an exudate has been thrown out which tends to hold the fragments in place. We have almost invariably found the antrum filled with blood in the comminuted fractures, so that the antrum drainage is a very important factor, as these blood clots almost invariably become infected. At the time of the operation a large antromental opening is made under the inferior turbinate to allow for drainage after the packing has been discontinued. In fractures of the frontal sinuses, if there is bleeding, the clot as a rule becomes infected and drainage is important. The posterior wall of the sinus or base of the skull may be fractured and a meningitis follow. A very careful observation of the sinuses should be made with a radiogram, if possible, and drainage instituted where there has been bleeding into the sinuses. In extensive fractures of the frontal, where the radiogram shows the frontal cloudy and there is a suspicion of fracture through the posterior wall, we believe external drainage of the frontal sinus is indicated.

The depressed malar bone may be elevated best by grasping it through the skin with a sharp hooked tenaculus forceps and bringing it into place. Elevation of the bone may be made in this manner much easier, as a rule, than by making an incision through the skin and introducing a hook or elevator, although this latter method is still used by some. Elevation can also be made through the antrum in some cases. An opening is made into the antrum as in a radical antrum operation, and through this a smooth blunt instrument may be used to elevate the bone. There is little difficulty in keeping the bone in place after ré-

ducing the fracture, as a rule, but the fragments may be wired if necessary. Where the zygoma is displaced outward, firm pressure is necessary over the prominence to reduce it, and it may be necessary to use continuous pressure from a wire attached to a headgear to bring about the gradual reduction.

Fractured nasal bones are pushed into place with a blunt elevator used intranasally, and if not comminuted too much will remain in place or can usually be held in place with an intranasal splint. We have seen little benefit from external splints in the majority of cases. If the bones are badly comminuted and will not remain in place a mold may be made of the nostrils intranasally with dental compound. To these molds wires are attached, which are bent over the nose and anchored to a headgear or if the superior maxilla has been fractured, may be attached to the appliance used in the mouth, thus holding the comminuted nasal bones in place. In lateral displacements of the nose, particularly where the nasal process of the maxilla has been fractured, it may be almost impossible to reduce the fracture and hold it in place, and in these cases external pressure can be applied to the side of the nose by attaching wires to the headgear as advocated by Dr. Kasanjian. Depressed comminuted fractures of the nose may also be elevated by putting a pin through the nose externally and anchoring this to the headgear. While we do not advocate external splints, we have found it advisable at times to make a light mold of dental compound which is placed over the dorsum of the nose, which will aid in holding the comminuted bones in place and give protection from without.

In many of these cases of extensive fractures of the face the absence of pain and discomfort is very noticeable, and often the patients show but little concern about the treatment. In extensive fractures of the superior maxilla and alveolus there is usually little pain in manipulating the parts so that the work can be done under local anesthesia. This is probably due to the anesthesia produced by the stretching or compression of the maxillary division of the fifth nerve. As has been stated, union takes place early in fractures of the facial bones, so that by the second or third week the bones are quite firmly united.

SEQUELAE.

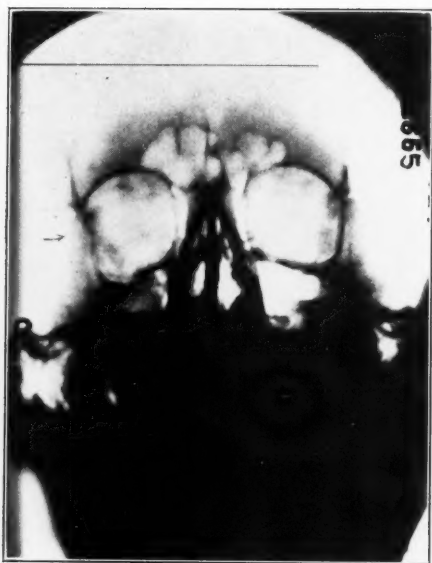
1. Numbness of the cheek may persist for some months and later be followed by a neuritis.
2. Malocclusion of the teeth frequently occurs.
3. Purulent dacryocystitis frequently develops.
4. Disturbance of vision due to displacement of the eyeball or injury to muscles or the optic nerve may follow extensive fractures.
5. Chronic sinusitis frequently develops.
6. Deformities of eyelids occur in some cases, due to scar contraction.
7. Sequestra may be extruded from time to time following the injury and their presence can be diagnosed, as a rule, by the continued swelling, redness and tenderness over the wall of the antrum.

REMARKS.

1. The frequent deformities existing after injuries to the face indicate that the rhinologist is not stressing this line of work.
2. Some of the nasal accessory sinuses are almost invariably involved in facial fractures.
3. Good cosmetic and functional results can only be obtained by early treatment, with proper reduction of the fracture and drainage of the sinuses when indicated.
4. A more careful study of the pathology of fractures of the facial bones and nose will, I believe, aid in the diagnosis, prognosis and treatment of these mutilating injuries.

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Orbital wall fractured inward and downward.

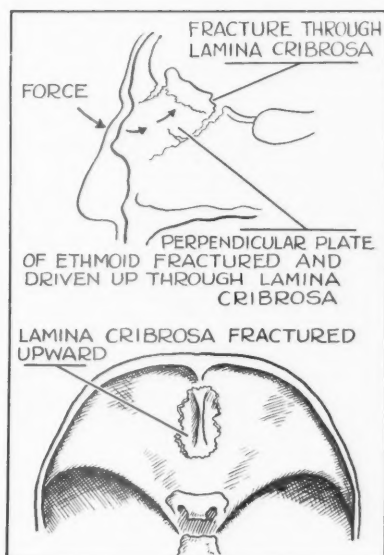
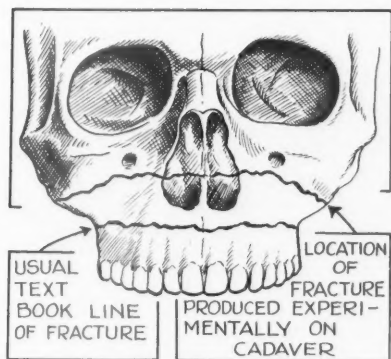


Depressed fracture of nose.





Headgear for holding fractured superior maxillary and nose.



XXXIX.

OSTEOMYELITIS OF THE FRONTAL BONE: TWO
CURED CASES.

By JOHN J. SHEA, M. D.,

MEMPHIS.

Osteomyelitis of the frontal bone is a progressive disease and its surgery must be timely and radical. Formerly it was a fatal condition, but with bolder cranial surgery successful cases are being constantly reported. Two cases included in this report were of boys, one twelve, and the other sixteen years of age, and were operated upon by Dr. R. E. Semmes.

The bacteriology of osteomyelitis of the frontal bones is the same as osteomyelitis of the long bones. This severe complication may follow any operation, simple or radical, on a suppurating sinus, or may occur spontaneous with the infection of the frontal sinus. Usually it occurs whenever the diploe is opened and exposed to an infection against which insufficient immunity has not been established.

The diploe is spacious in the growing skull, which makes the youthful frontal bone more susceptible to a spreading infection.

Osteomyelitis occurring immediately after an operation is more likely to be serious and rapid in its destruction than when it does not appear for ten days or more. Incision at the fluctuating point over frontal osteomyelitis is of little or no value, and the surgical attack must be formable and extend beyond the limits of the necrosis into the healthy bone. When possible both tables should be removed as a trench beyond the diseased bone to prevent its spreading. Though necrosis starts within the frontal sinus, the first external manifestation may be high up on the forehead, for the infection travels by way of the diploic space. This mode of spreading is the only reason why the point of internal rupture is most frequent at the former site of the anterior fontanelle, as occurred in both of our cases. The infection spreads more readily over the vault than

back into the base of the skull, and fortunately the barrier to its progress can be easily made on the vault. The operative incision should be free and without regard to subsequent deformity, for the pathology is serious. We have found that local is better than general anesthesia in these skull cases, and if properly administered can be used in young subjects; our second case was only twelve years of age.

CASE REPORTS.

Case 1.—History: C. B., age 16, male, was admitted to the Baptist Memorial Hospital on July 10, 1923, in a semistupor. Five weeks previously he had gone in swimming and contracted an acute sinusitis limited to the right group of sinuses. The patient was treated with suction by Drs. Culley and Guyton, of Oxford, Miss., for two weeks, during which time the discharge was purulent. Simultaneously with the stopping of the nasal discharge there appeared an inflamed swelling over the right frontal sinus. Under ice packs this swelling receded but subsequently became worse and extended up to the hairline. The greatest degree of fluctuation occurred at the hairline over the site of the anterior fontanelle, and an incision was made at this point. The patient did not improve at home and was transferred to the Baptist Memorial Hospital, at Memphis. Upon examination the lad was mentally dull, his nasal cavities were clean and the skin over the right half of the forehead was swollen and inflamed with a small incision at the hairline. The urinalysis was negative, the white blood cells were only 7,500 with polys 57, small lymphocytes 34, large lymphocytes 7, and eosinophiles 2. The roentgenogram showed an extensive osteomyelitis of the right half of the frontal bone with a communicating tract leading down to the right frontal sinus.

Therapy: Dr. R. E. Semmes, under local anesthesia, turned back a scalp flap and exposed the involved area. The outer table of the right and middle thirds of the frontal bone presented a moth eaten appearance and was removed. There was a collection of unorganized inflammatory material between the skin and the fascia, with a flat sequestrum to the right of the midline. The outer and inner tables had been destroyed over the former site of the anterior fontanelle and an extra-

dural abscess was evacuated. A rubber tube was used for drainage of the abscess. (Fig. 1.) The outer table was removed down to the right frontal sinus, and a rubber drain was passed down into the nose from the sinus. The flap was sewed back into position and the patient reacted with a rise of temperature. His cerebation improved, and at the end of a week the right anterior and posterior ethmoid sinuses were opened and curetted, as the anterior cells were filled with granulations and polyps. After four weeks of drainage he became dull again and then semiconscious. Dr. Culley drained the extradural abscess, and the lad made an uneventful recovery. The patient is still enjoying good health at the end of four and one-half years. (Fig. 2.)

Comment: The patient was a young lad with a growing skull, and the onset of the osteomyelitis was independent of any surgery. The infection traveled upwards through the diploic space to rupture inwardly at the former site of the anterior fontanelle.

Case 2.—History: J. W. Jr., male, 12, presented himself on August 14, 1925, to Dr. T. E. Huey of Anniston, Ala., with an acute inflammatory swelling over the left frontal sinus. Two years previously, while playing, he ran into a brick pillar which rendered him unconscious. There was considerable injury to the soft tissue of the forehead, but an examination failed to discover any injury to the frontal bone. Following this accident the lad began to have frequent headaches and upon contracting an acute summer cold he rapidly developed an acute frontal sinusitis. Coincident with the onset of the acute frontal sinusitis there developed an acute inflammatory swelling over the left frontal sinus. A roentgenogram showed the frontal bone to be intact. (Fig. 3.) An external frontal sinus operation was performed by Dr. Huey, and drainage established down into the nose. The external wound was left partially open for drainage. Five days later surgical intervention was necessary on the right side for a similar condition. The external swelling continued, and on the fourth day after the second operation drainage was established in the midline, but the osteomyelitis spread in all directions.

Examination: When he was admitted to the Baptist Memorial Hospital, at Memphis, on October 8, 1925, there was

found a swelling over the whole forehead with a discharging central wound. The urinalysis was negative. The white blood cells were 7,900, with polys 70, small lymphocytes 24, and large lymphocytes 6. A roentgenogram taken at this time showed considerable destruction of the frontal bone, with a loss of the inner table for an area of 1 x 2 inches at the former site or the anterior fontanelle. (Fig. 4.)

Therapy: Dr. Semmes made a wide incision and turned down a scalp flap exposing the frontal bone. The outer table was extensively removed and the diploic space was found to have been destroyed and its position occupied by pus and granulations. The inner table had been lost for the size of a silver dollar at the former site of the anterior fontanelle, and an extradural abscess was drained at this site. The frontal sinuses were entered from above and their mucous membranes curetted thoroughly with a hope of obliterating the sinuses. The external wound was close, leaving lateral drainage. The wound gradually healed, and on May 11, 1926, a roentgenogram showed reformation of new bone and the frontal sinuses to have continued their growth. The patient has had no further trouble and has progressed favorably in school.

Comment: The patient was also a young lad with a growing skull, and its onset was independent of surgical interference. I do not think the blow on the forehead two years previous predisposed the bone to a spreading infection.

COMMENT.

Occurrence: The disease may follow any operation of an acutely infected sinus or may arise as a complication in unoperated cases. It has complicated operations of masters and novices.

Roentgenography: The taking of roentgenograms during the course of the disease will furnish the best record of the progress of the pathology and the rapidity of the postoperative bone reformation.

Therapy: (a) "You may know a bone surgeon by his chips." This applies to the surgery of frontal osteomyelitis. It is better to sacrifice a suspicious area than to leave it. (b) Intravenous medication is of value. McKenzie recommends the use of colloidal silver, and though we did not use it or our American

mercurochrome, I would not hesitate to try it, should one of our future cases begin to recede after surgery. (c) Vaccines—They are of little value during the acute stage but may be used during the later stage when the immunizing processes become sluggish. The same may be said of foreign protein therapy.

Regeneration: If the inner table can be saved the bone will rapidly reform in the young.

LITERATURE.

McKenzie, Dan, in the *Journal of Laryngology and Otology*, Vol. 28, Page 6, 1913, gave a very exhaustive report of the cases up to that time and in the May issue of the same journal during the past year has reported five more cases.

Bulson, A. E., read before the American Academy at Chicago, in 1925, a very full report of the American and foreign literature upon the subject, and reported a personal case.

Hastings, Hill, read before the American Laryngological Association, May 23, 1927, a case report of an acute fulminating osteomyelitis of the frontal bone of possible hematogenous origin (dental focus). This paper appeared in the December issue of the *Archives of Otolaryngology*, Vol. 6, No. 6.



Fig. 1. Case 1.—Roentgenogram showing tube draining extra dural abscess.



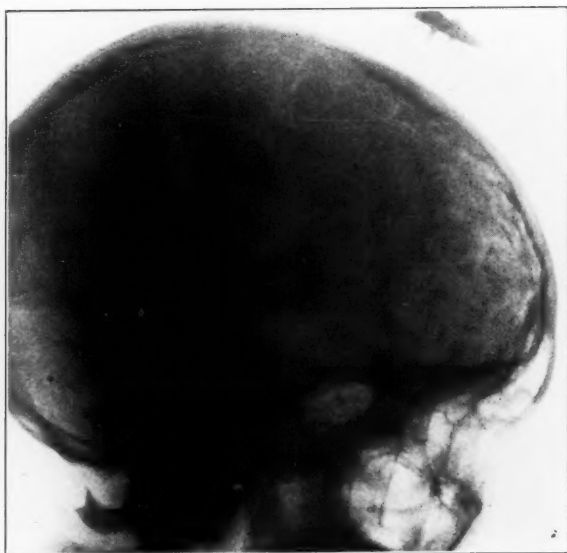


Fig. 2. Case 1.—Roentgenogram showing the reformation of the frontal bone and the frontal sinus.



Fig. 3. Case 2.—Roentgenogram showing the beginning of the osteomyelitis.





Fig. 4. Case 2.—Roentgenogram showing how the osteomyelitis spread up through the diploe to perforate internally at the former site of the anterior fontanelle.

XL.

OSTEOMYELITIS OF AN INFANTILE SUPERIOR MAXILLA.

By JOHN J. SHEA, M. D.,

MEMPHIS.

Osteomyelitis of the superior maxilla in nursing infants is rare and usually misleading. The external manifestations are orbital in character and present the signs and symptoms of an acute suppurative dacryocystitis or ruptured ethmoid abscess. The incision should not be made external because of future deformity. The case reported below is a good example of the folly of orbital incisions and of the horrible after effects. The incision of choice is located in the sulcus of the cheek, and if an area of fluctuation is present should be over same. The following case was very interesting and baffling.

History: G. G., male, age 4 months, was referred by Dr. E. C. Ellett for examination and treatment. At the age of two months the left side of the face became swollen, and the lower lid of the left eye edematous and reddened. There was a local inflammatory swelling in the upper left gingivolabial sulcus with a corresponding area of the gum involved. An incision in this area freed a tooth bud from its nest and the inflammation partially subsided. The orbital swelling increased, so an incision was made into the outer quadrant of the left orbit with no result. The two incisions continued to drain pus and a second tooth bud was spontaneously discharged. After two months of the above the child was referred to Dr. Ellett.

Examination: The child had, on admission to the hospital on March 2, 1926, an orbital cellulitis with a discharging sinus in the outer lower quadrant of the left orbit. The left naris was filled with a purulent discharge, and a discharging sinus of the left superior maxilla made its exit into the sulcus of the cheek. Necrotic bone could be felt at the bottom of this sinus. The cavity of the left maxillary sinus was very small and filled with pus. An external incision into the ethmoid cells

showed the cells to be filled with a purulent discharge and their lining membrane was thickened. The above surgery was of no value. A small piece of the superior maxilla taken for biopsy, through a gingival incision, was reported as having "chronic inflammatory changes." Six weeks later a radical antrum operation was performed, and radium seeds implanted into the cavity, as we feared that the condition might be a sarcoma. The child gradually improved, though the gingival incision continued to drain, and eighteen months later a sequestrum was removed which contained a semiformed tooth. The child had normally grown in size and mind, but the external incision in the outer angle of the orbit had produced a disfiguring scar.

Comment: This case was originally an osteomyelitis of the superior maxilla, and the orbital cellulitis was the result of the incision in the external angle of the orbit. I am now convinced that the external incision into the ethmoids could have been avoided if the osteomyelitis had been radically attacked through the gingival incision.



Fig. 1. Photograph made after the orbital incisions had healed.



Fig. 2. Photograph taken May, 1927, showing the deformity caused by the external incision.

XLI.

LOCALIZATION AND DRAINAGE OF BRAIN ABSCESES OF OTITIC ORIGIN.

By JOHN B. POTTS, M. D.,

OMAHA.

Brain abscesses of otitic origin are by far more numerous than from all the other sources combined. Kopetzky gives the following: Körner, in an analysis of 119 cases, found temporal 79, cerebellum 40. Barr, in 75 cases, found temporosphenoidal lobe 55, and cerebellum 13. The observations of Heimann, Tod and Neumann are in close accord with the observations of Körner, namely, that the temporosphenoidal lobe is the site of the abscess in approximately 65 per cent of the cases. Eagleton says: "In my series, excluding all truly traumatic cases, out of the first 44 brain abscesses, 40, or over 90 per cent, belonged to the secondary adjacent types, chronic or acute. The vast majority of cerebellar abscesses originate from aural infection. In 117 recorded postmortems analyzed by the author, the abscess was of otitic origin in 99 cases." If we add to this abscesses of nasal origin it is apparent that the otorhinologist is more directly concerned with brain abscesses than all the other branches of medicine combined.

The above statements lay a heavy burden on us, as the recognition, drainage and postoperative care of a brain abscess is a serious responsibility. The condition is one requiring surgical intervention or the patient will die. The diagnosis and location of the abscess is frequently exceedingly difficult, and operative and postoperative care are so fraught with danger that the mortality rate is high. Lund,¹ in his recent article, states that the reported statistics from several large otologic clinics are as high as 75 to 80 per cent mortality, and from his own report of the Municipal Hospital at Copenhagen, he says, "we succeeded in curing 10 out of 54 patients with otogenous brain abscess." He, however, states that his success with his later cases has been much better than with the earlier ones. This, I think, with the increased attention given to the subject

and the improvement in surgical technic, is becoming generally true. Eagleton reports a mortality of 75 per cent, and quotes Mygind, 42 abscesses of otitic origin with only four recoveries, or over 90 per cent mortality, and Niëhsman, temporosphenoidal lobe, $33\frac{1}{3}$ per cent recoveries, and cerebellar lobe about $10\frac{1}{2}$ per cent recoveries. King² reports four consecutive cases with no fatalities. During the year 1927, Dr. Callfas and I had five cases with four recoveries and one death. Two were temporosphenoidal, no fatalities; 3 cerebellar, 1 death. One case was an adult and four were children. Two occurred in the same family.

DIAGNOSIS.

The presence of a discharging ear or history of a recurrent discharging ear in a patient with any symptoms suggestive of an intracranial lesion, should direct our attention to serious consideration of a brain abscess. If a mastoid operation has been performed and the patient fails to make a satisfactory recovery, the possibility of an intracranial lesion must be excluded. If a patient, apparently doing well, has a chill or an unexplained rise in temperature, it may be the evidence of an initial invasion of the cranium.

HISTORY.

A careful history will often elicit important data. It may be that for several weeks there has been a loss of the feeling of well being, a sense of anxiety, of something wrong, inability to concentrate on work, occasional inability to speak certain words, a spell of so-called indigestion with vomiting, loss of weight, transient blindness, or weakness of an arm or leg and practically always headache.

In one of our cases, an Indian, although he had had discharging ears for several years and had not worried about them, became anxious about himself, and of his own accord came down from the reservation and went to the Methodist Hospital, asking for treatment. Another time, a student came in for glasses on account of headache and occasionally spells of dimness of vision. The history disclosed that he had a mastoid operation five weeks previously and that for the past week he had noticed inability to name certain words. He had a left temporosphenoidal lobe abscess.

General symptoms: Otitic or nasal sinus infection; debility; general malaise; headache; fever; chills; vertigo; vomiting; visual disturbance, optic nerve palsies, choked disc; lowered cerebration; slow pulse; subnormal temperature; convulsions; pulse pressure; blood pressure; crying out in sleep; spinal fluid; blood picture; odor.

Given a patient with a focus of infection and two or three of the above symptoms, a brain abscess must be considered. Headache is an almost constant symptom. There is often a distinct history of onset with a little chill or fever, after which the patient has never felt quite well. The vomiting has frequently led to a false diagnosis, and time has been lost treating the stomach.

Slow pulse, "bradycardia," is a very reliable symptom. It is more liable to be present in brain abscess than in any other intracranial lesion. It must be sought for carefully, as it frequently occurs only for an hour or so in every day or two or three days, until in the late stages. A two-hour pulse record, counting a full minute, is desirable. The night record is frequently the most valuable. If only one-fourth minute is counted, a mistake of four beats above or below sixty is easily made, and the difference between 56 and 64 is frequently sufficient to decide a tentative diagnosis for or against a brain abscess. A convulsion associated with a focus of otitic infection is almost positive evidence of an intracranial lesion.³ The blood pressure in acute abscesses rises. The pulse has a full, firm beat. Subnormal temperature. Except at the initial stage, the temperature, contrary to the usual rule of infection elsewhere in the body, is normal or subnormal.

Spinal fluid: Increased pressure and cell count, but usually sterile. Blood, only moderate increase of white blood cells.

Odor: A peculiar odor and profuse sweating may precede death.

Roentgen ray: Does not ordinarily show an abscess.

Localizing symptoms: Temporal lobe, active labyrinth, aphasia, word sound, auditory memory, papilledema, muscle spasm of opposite side, muscle paresis of opposite side, homonymous hemianopsia, partial deafness, dilated pupil of same side, ptosis, pain over temple, tendon reflexes, Babinski, Oppenheim.

An active labyrinth is evidence of temporosphenoidal lobe abscess, only as a negative evidence against a cerebellar abscess, the latter being most often associated with a dead labyrinth.

Aphasia is a very valuable evidence of left temporal lobe involvement in a right-handed person, and vice versa. The word and memory centers are unilateral and located in the first, second and third temporal gyri, respectively.⁴ As the pressure from an abscess is not constant, aphasia, especially in the early stages, when it is most important, is often transitory. This is true of practically all the early evidence, and thus frequent careful examinations and intelligent nursing is most important if a much desired early diagnosis is to be made.

Papilledema is a much heralded symptom and is responsible for many gravestones, as it frequently either does not develop or else is very late, and the surgeon waiting for the classical symptoms sees the exit of his patient.

Paresis of the opposite side from the suspected tumor is valuable and fairly reliable evidence of temporal lobe abscess. It is usually present in the face first, then in the arm, and the leg is the last to be affected.

Homonymous hemianopsia is often present fairly early and is a very important localizing symptom of temporosphenoidal lobe lesion. It is due to an involvement of the association fibers running from the cortical optical center in the cuneus to the geniculate bodies, Myer's tract, which passes through the temporosphenoid lobe.⁵

Dilated pupil, ptosis, pain over temple, occur on the same side. Increased tendon reflexes. Babinski's and Oppenheimer signs may be present.

Localizing symptoms: Cerebellar; inactive labyrinth; thrombosis of the lateral sinus; static imbalance, incoordination ataxia; pass pointing, finger, finger to finger, finger to nose, toe, up and down; papillary edema; adiadokokinesis; posture; vomiting; nystagmus; vertigo; pressure on cranial nerves; transitory nature and variability of signs; crying out in sleep; occipital headache.

The most common path of infection of the cerebellum is through the labyrinth. As I stated before, a dead labyrinth points to a cerebellar abscess.

Static imbalance: The cerebellum is the coordinating center of the brain and, therefore, normal muscle movements are liable to be disturbed. The unsteady gait, inability to stand on one foot, tendency to fall, exaggeration of movements, inability to synchronize, the pronation and supination of the two hands when done rapidly (adiadokokinesis) are all evidence of incoordination.

Vomiting of the projectile type, nystagmus, especially vertical and extreme vertigo, are all late manifestations of cerebellar abscess.

Lund²: "A review of the literature, as well as of our own cases, shows that one significant cause of so many otogenous brain abscesses not being diagnosed or, at any rate, not evacuated, is just this, that the symptoms present in each case—nystagmus, paresis of extremities, aphasia, mispointing, etc.—or the operative findings on chiseling the mastoid process and middle ear have suggested an abscess of the temporal lobe, where the accumulation of pus then was sought to no avail, while autopsy proved it to be located in cerebellum, or vice versa. This alone even justifies the extensive use of the diagnostic puncture of the brain as an indispensable aid in cases where abscess of the brain is the only condition possible; and in this manner we have repeatedly succeeded in revealing an abscess of the temporal lobe, when beforehand we expected a cerebellar abscess, and vice versa." Papillary edema is more often present in cerebellar than in temporal or frontal lobe abscess.

TECHNIC OF THE OPERATION.

The five cases I am reporting were done by the same technic. All followed mastoid operations. The exposure of the dura, done by an extension of the incision from the mastoid wound. The cerebellar abscesses are drained from below and back of the lateral sinus and the temporosphenoid by uncovering the dura above the ear. In each case bone is removed so as to expose an area about 4 cm. in diameter. A small incision is made through the dura but no dural flap. If the abscess is definitely located, the Gifford brain abscess seeker is at once

passed into the abscess; if the localization is not definite, a large blunt needle with canula is used. When the abscess is located, the Gifford seeker is introduced by the side of the needle, and the needle withdrawn. The blades of the seeker are then gently separated by pressure on the handle until a rubber tube of about one-half inch in diameter can be passed between the blades into the abscess. The seeker is then removed, leaving the tube in situ. The tube is anchored by splitting the top into three or four sections and stitching these to the edges of the wound. Iodoform gauze packing over the dura beneath the flaps of the tube and a large 1/5,000 bichlorid moist dressing completes the dressing. The dressing is kept moist by adding bichlorid solution every four hours. The dressing is changed daily and the pus removed from the tube with a pipette. The silk stitches in the tube are touched with a 4 per cent silver nitrate solution at each dressing. On the fourth to the sixth day, or as soon as the drainage tract is well established, this tube is replaced by a "U" shaped silver wire made of fine silver 2 mm. in diameter. The ends of the wire are left about three-quarters of an inch long and bent so as to lay smoothly over the edge of the wound. The wire loop should be gently manipulated each day to keep the tract open to the abscess, and is not removed until the drainage has entirely ceased. Finally, when all evidence of infection is gone, the skin is elevated and the wound closed. Swelling of the brain is liable to occur after the introduction of the tube. This can usually be controlled by intravenous injections of hypertonic glucose or sodium chlorid solution, or, if slight, by active catharsis.

The brain seeker, devised and described by Dr. Harold Gifford,⁷ is shown in the illustration. The double blades are accurately approximated so that the point makes a clean entry. The inner surface of each blade is slightly hollowed to allow a freer egress of pus, and the blades are crossed so that they spring together firmly and are opened by pressure on the handle. The blades are marked in inches. It has two important advantages: First, it gives a positive diagnosis of pus when the abscess is entered, and second, the drainage tube is introduced into the abscess between the blades, thus making its introduction and position sure and without trauma. The whole

procedure only requires the passage of one instrument through the brain and greatly reduces the danger of infecting the brain. It avoids the danger of losing the abscess.

CASE REPORTS.

Case 1.—Mr. W. R., age 19. Entered the Methodist Hospital October 5, 1926, on account of discharging ears.

History: Right ear has been discharging for six years, left ear for two years. The left ear never has been as bad as the right. Has been treated by different doctors without benefit. Decided to come to hospital for operation. Has never had much headache, no nausea or vomiting.

Patient is medium size, rather thin, mentally alert. Temperature 98.6, pulse 72, respiration 18. Fairly profuse purulent offensive discharge from the right ear, not much from the left. He was put on conservative treatment for the ears and held for observation. For about two weeks he ran a fairly normal course, but had an occasional day with 1 to 1½ degree of temperature and a pulse rate from 64 to 80. On the morning of the twelfth day, October 17th, he complained of dizziness, and on the following day, October 18th, he had a headache over the left eye and was dizzy. The discharge from the left ear stopped. Temperature 99.6, pulse 70 to 90. October 19th, he was nauseated, with a temperature ranging from normal to 100. October 20th, he was up and around, but had a little headache and vomited twice. October 21st, felt better during the day but at about 10 p. m. became quite sick, was semidelirious and had a severe vomiting spell. October 22nd, stuporous and mentally very dull. The right ear was dry, the left had a moderate amount of purulent offensive discharge. Has been vomiting and complained of dizziness and headache. Temperature 98, pulse 60. W. B. C. 10,500, P. 80, S. L. 20.

General examination negative. Probably has intracranial lesion. (Dr. Bliss.)

Eyes normal. Fundus normal. (Dr. Patton.)

Neurologic: Mental dullness, approaching stupor. Pain in left temporal region. Sensory aphasia, type Alexia. Cannot name pin, dollar or handkerchief. There is also a suggestion of paraphasia. Makes a few mistakes in reading. Miswrites "fine" in this is a fine day.

Diagnosis: Brain abscess, left temporal lobe, posterior part. Recommend operation at once. (Dr. G. A. Young.)

An operation was done at once. The mastoid was eburnated, no cells; fairly large cavity back of the antrum, filled with caseous offensive pus. Incision extended up and dura exposed above antrum. Vertical incision through dura and Gifford brain abscess seeker passed into posterior portion of the temporal lobe and the abscess located at depth of one inch. About one ounce of thick pus with greenish tinge evacuated. Rubber tube three-eighths of an inch in diameter introduced between blades of instrument into abscess. Outer end of drain split longitudinally into four parts, making four flaps three-fourths of an inch in length. These flaps were then stitched to the skin in the four sides of the wound with silk. This held the tube gently but firmly in position.

The drainage for the first three days was profuse and then decreased rapidly, and on the fourteenth day (November 5th) after the operation, there was no drainage and the wound was clean. The rubber tube was removed on the fourth day and replaced by a U-shaped fine silver wire, 2 mm. in diameter. At each dressing this wire was carefully manipulated, just sufficiently to prevent the brain from uniting between the wires. After the tenth day the wire was gradually shortened, and on the sixteenth day (November 7th) it was removed. There were no complications, and he was discharged November 18th.

Case 2.—Mary P., age 10 years. November 21, 1927. Carried into Methodist Hospital.

History: Three weeks ago had the flu. Pain in right ear began in a few hours. Vomited, very little nausea. Ear began discharging on the fourth day and she felt a little better for a short time. Then frontal headache developed, and again began vomiting. This has continued until now. Referred to hospital with diagnosis of suspected brain abscess.

General examination negative. Right ear discharging. Left ear, old perforation, dry.

Neurologic: Mental condition bright. Facial movements symmetrical. Dis-adiadokokinesis present in right hand. Pointing with right leg more unsteady than left. The ataxia in both leg and arm is not marked. Left ankle jerks seem more ex-

citable and active than right, with a suggestion of clonus. (Dr. Young.)

Eyes: Choked disc $2\frac{1}{2}$ diopters, bilateral.

The above symptoms point to a right cerebellar localization. The following negatives contraindicate a right temporal lobe abscess: (1) Absence of mental hebetude; (2) absence of hemianopsia; (3) absence of loss of sensory discrimination; (4) absence of left sided paralysis or weakness; (5) absence of Kernig and nystagmus; temperature 98—97.4; pulse 64—72. Laboratory: W. B. C., 15,300; P., 60; S. L., 40. Urine normal.

Operation, March 23, 1927. Bone very hard, upper half of mastoid sclerosed. Cavity in lower half filled with pus. Sinus wall eroded and covered with granulations. Sinus exposed to knee, apparently not thrombosed. Cerebellum exposed back and below sinus. Wound packed for 24 hours. March 24th, cerebellum explored with aspirating needle. Abscess located at depth of $1\frac{1}{2}$ inches. Gifford brain abscess seeker inserted into abscess and about two or three drams of pus evacuated. Rubber tube inserted as described above. Some headache and vomiting for three days and then began to improve rapidly. No vomiting after the fourth day. Discharge of pus fairly free for two days and then lessened rapidly. On the sixth day there was only a slight drainage, and the tube was replaced by a silver wire. After April 2nd she had no more headache and made a rapid convalescence. The headache was apparently due to an edema of the brain and was relieved by active catharsis (magnesium sulphate).

Case 3.—John H., age 12 years. June 19, 1927. History: Had mastoid operation for an acute right mastoiditis in March, 1926. Ear has never quit discharging. Discharging fistula in mastoid. Some headache. Apparently in good health—attending school.

Operation, June 20, 1927: Mastoid reoperated upon. Some dead bone, pus and granulation back of antrum.

Following the operation he did not run a normal convalescence. Headache every day and a temperature of 99 to 101. On July 1st and 2nd his pulse and temperature were normal, but he still had a headache and did not want to get up. On July 3rd he vomited and his pulse was 60, temperature 98. On July 4th he was much the same, except that his pulse remained

above 70. On July 5th apparently about the same, except pulse 52. Headache.

Neurologic: Patient appears weak. Cooperates well in examination, though with evident effort due to weakness. Mentality clear. No clouding of consciousness. Hyperalgesia of right side of face. Lower middle type of facial weakness on right side. Hand grips equal. Dis-adiadokokinesis, right. Knee and ankle jerks all present. Babinski absent, bilateral. No loss of power in legs. Abdominal reflexes normal. Condition suggests abscess of right cerebellar hemisphere or subcortical abscess over right hemisphere of cerebellum. (Dr. G. A. Young.)

Eyes: Fundus, veins swollen. No choked discs. Fields normal. Horizontal and vertical spontaneous nystagmus present.

Operation: Dura exposed back and below knee of sinus. Abscess located with needle and drained as in previous cases.

After the operation he did fairly well for two days; then his temperature went up to 104 and his condition became steadily worse and he died on the fourth day.

Postmortem report: Brain was removed by the usual procedure. After removing the dura, the pia was found to be somewhat reddened. There was no exudate any place on the surface. An abscess cavity measuring about $1\frac{1}{2}$ cm. in diameter was found in the right lobe of the cerebellum. This had been completely drained through an opening in the calvarium. There were no other abscesses in other parts of the brain. Mastoid cells had been completely removed. Another opening into the calvarium above the ear entered the middle fossa.

Diagnosis: Abscess of the cerebellum.

Immediate cause of death: Septicemia, probably medullary embolis. (Dr. F. W. Niehaus.)

Case 4.—Max T., age 8 years. April 29, 1927. Brought into Methodist Hospital in coma.

History: Last fall had scarlet fever, followed by an acute otitis media and mastoiditis. A mastoidectomy was done, and he apparently entirely recovered. Three weeks ago he suddenly began to have fever—103 to 104—and vomited. Right sided headache.

Ten days ago had a lumbar puncture. Report, increased pressure, cells 25. Two days ago had choked disc, double

vision, and was stuporous. The next day he had a right hemiplegia, and the left side in clonic contraction. Last night was in a coma.

Physical examination negative.

Neurologic: Abdominal reflexes present, equal. Babinski, bilateral. Ankle clonus, bilateral. Knee jerks present. (Dr. G. A. Young.)

Eyes: Pupils unequal, right larger. Choked disc, R. E., 4 D.; L. E., 3 D.

Blood: W. B. C., 7,900; P., 62; S. L., 38.

Hearing: Not possible to obtain. No discharge from either ear.

Nose and throat normal.

Diagnosis: Brain abscess, otitic origin. Location probably right temporal lobe.

Operation, April 30, 1927: Reopened old mastoid. Some granulations around antrum. Dura, above and back of antrum exposed—covered with granulations. Lateral sinus exposed from knee downward five-eighths of an inch, apparently normal. Dura exposed up and forward $1\frac{1}{2}$ inch by 1 inch. Abscess located in posterior part of temporosphenoidal lobe.

Drained as in previous cases.

Culture: Pneumococcus.

Rubber tube removed May 4th. Wire drain removed May 11th. No further drainage. Recovered.

Case 5.—Franklin P., age 7 years. June 15, 1927. History: Several months ago had some pain in the right ear. Two weeks before coming to the hospital had a recurrence of the pain with a chill. This four days before coming to the hospital. Also had another chill the day before coming to the hospital, which was followed by high fever. Ear discharging.

Operation performed the same day. Simple mastoidectomy. Mastoid filled with a bloody fluid, the antrum and cells adjacent to the antrum filled with pus. Uncovered three-fourths inch of sinus below the knee. Bloody fluid escaped from around the sinus. Sinus covered and mastoid packed with bichlorid gauze.

Following the operation the temperature made daily excursions from normal to 105. This was probably present before he entered the hospital. The white blood count remained about

the same. He had a chill on the second postoperative day. Cultures from the mastoid, hemolytic streptococcus and staphylococcus. General examination negative.

On the second day the right jugular vein was tied and the sinus exposed from the tip of the mastoid to two inches back of the knee of the sinus. The sinus appeared normal here and bled freely when punctured. It was blocked with a pack of iodoform gauze, placed between the sinus wall and the cranial wall, and then the exposed sinus was laid open and the contents evacuated. About the knee the wall was broken down and draining pus. After this he had one more rise in temperature the following day, and then ran a fairly normal course for about a week.

The blood culture on the 20th and 21st grew staphylococcus albus. The W. B. C. on June 16th was 17,000; 17th, 13,500; 18th, 12,000; 20th, 17,800; 24th, 19,300; 25th, 16,000. Pulse, on the 19th to 22nd inclusive, dropped to about 65 in the late afternoon of each day. The child did not do well, but we ascribed this to the infected sinus. On the 20th, the discs were a little blurred and the wound continued to discharge. Had pain in right side of head. On the 27th, had pain in right ear. Some jerking of legs, left worse. Exaggerated knee jerks. Ankle clonus, right. Ocular findings not recorded.

Diagnosis: Suspected brain abscess.

Exploratory operation: Pus coming from brain, apparently from fistula under necrosed wall of lateral sinus just back of knee. Fistula probed and abscess located at depth of about three-quarters of an inch. Drained with wire as described above. Another small abscess was located extradural above the root of the zygoma. Following this the child made an uneventful recovery and left the hospital July 24th.

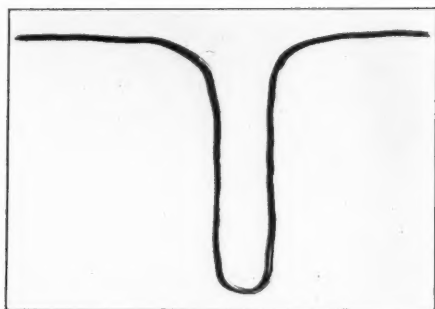
In this case the thrombosed lateral sinus masked the brain abscess. After the sinus was controlled, the second rise in temperature probably was due to the formation of the extradural abscess, which again obscured the picture. In spite of the temperature at this time, 104.6 on the 23rd at midnight, and 103.4 the following night, the pulse remained comparatively low, not going above 100, so that the pulse and headache were the outstanding symptoms.

We have kept in touch with these four cases since the operation. There has been no recurrence, and they are well and living at the present time.

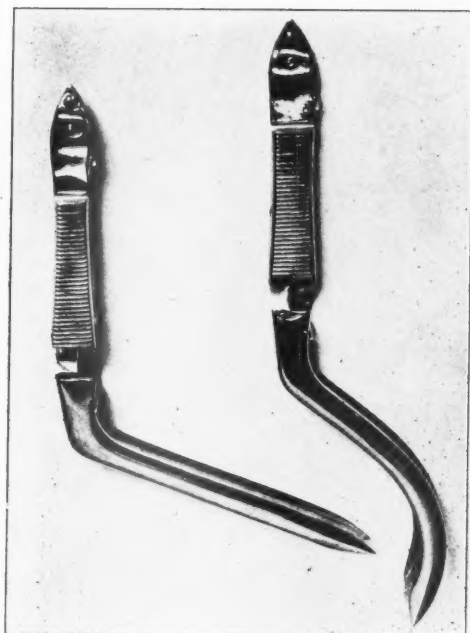
We are indebted to Dr. G. A. Young for his able assistance in the neurologic examinations and diagnosis, and to Dr. W. B. Moody and Dr. F. W. Niehaus for the laboratory reports.

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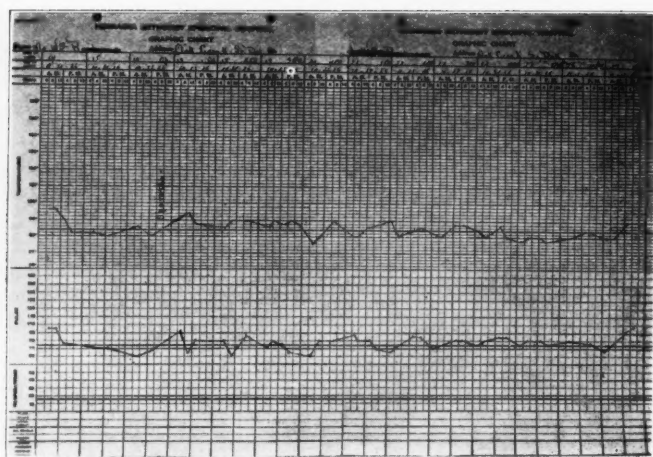


U-shaped wire drain.

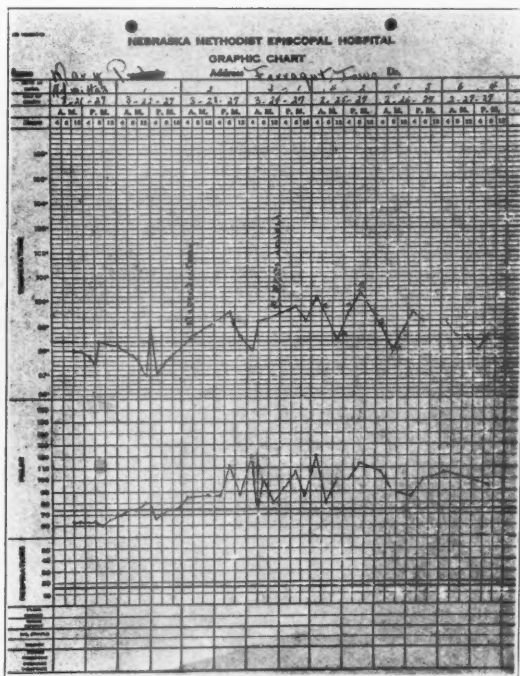


Gifford Brain Abscess Seeker.





Case 1.

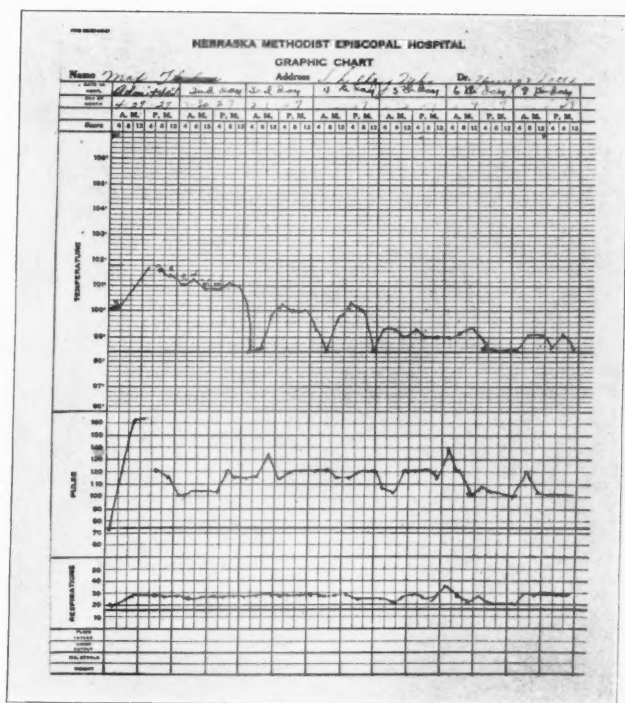


Case 2.



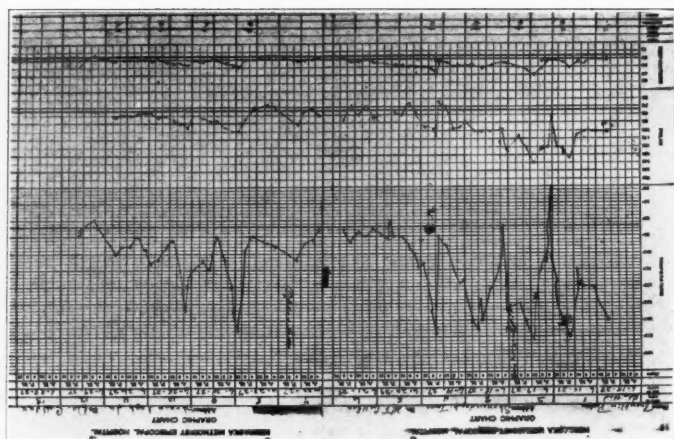


Case 3.



Case 4.





Case 5.

XLII.

EXPERIENCE WITH A NEW MASTOID FLAP AND DRAINAGE (ELLIS).

By BENTON N. COLVER, M. D.,

BATTLE CREEK, MICH.

In September, 1926, Dr. C. Ward Ellis presented a series of postoperative mastoid cases before the Ophthalmological and Otolaryngological Section of the Michigan State Medical Society. The patients shown at that time were to illustrate a new approach and a method of drainage and postoperative care that had been used by him and his associate, Dr. Heckert, for about a year. In all, to date (February, 1928), about 50 of these cases have been handled by them.

The histories and the postoperative appearance of the patients shown by Dr. Ellis, together with his enthusiasm for the method, led several men throughout the state to try it. In our clinic three surgeons have followed his technic in the few cases that we have had in the past sixteen months. Our experience leads us to believe that its value justifies this report with the hope that a much wider clinical trial will result. To the best of our knowledge, this technic is original with Dr. Ellis, having been evolved by him during the past three or four years. The procedure is as follows:

1. *The Skin Incision* (Plate I).—In preparation for the operation the hair is shaved from an area extending at least three inches behind and two and one-half inches above the tragus. Instead of the usual curvilinear incision through skin and periosteum, a short distance behind the attachment of the pinna, a much wider incision is made, and at first through the skin and subcutaneous tissue only. (Plate II.) The proposed line of skin incision is outlined with the back of the knife-point, and scrutinized carefully before making the actual incision. Five or six cross incisions are made to insure correct closing coaptation. The incision begins at the top of the attachment of the pinna, passes directly backward for about one and

one-half inches, and with a short curve continues vertically downward nearly to the level of the tip of the mastoid. This posterior line is approximately two inches posterior to the tragus. Again with a short curve the line is turned horizontally forward across the tip. The incision, as will be seen, lies for the most part, within the hair line. In our technic we turn the incision forward a bit sooner so that the final line does not pass horizontally but rather at a slight angle downward to and across the tip. This will be noted in the patient to be shown you and in the slide of that case from our clinic. This we feel avoids entering the soft tissue, where it is quite thick, just anterior to the posterior inferior angle of the Ellis flap.

2. *The Skin Flap* (Plate III).—After this first incision the skin flap is elevated and dissected inward toward the ear from all borders until it is lifted about three-fourths of an inch from its posterior free edge and a gradually lessening distance as one approaches the ends of the incision. This uncovers the areolar tissue overlying the periosteum, which is at this stage uncut.

3. *The Periosteal Incision*.—An incision is now made through the periosteum, beginning above at the point of the skin incision and ending below also at the exact point of the skin incision. The periosteal incision, however, diverges from the line of the skin incision immediately on starting backward and is about one-half inch inside of the skin incision as it passes vertically downward.

4. *The Periosteal Flap* (Plate IV).—After this incision the posterior periosteal border is loosened and elevated backward for a short distance. The bone is then uncovered from above downward and forward by elevating first the periosteal border and then the whole thickness of the skin and periosteum as in the usual mastoid exposure. (Plate V.) Anterior to the periosteal incision is thus formed a short apron of periosteum which makes the periosteal suturing easier. As the elevation approaches the tip, scissors are needed to complete the dissection. (Plate VI.) On account of the breadth of the wound, no mastoid retractor was found satisfactory until the Andrews self retaining retractor was modified by making the slide arm to measure $3\frac{3}{4}$ inches instead of $2\frac{3}{4}$ inches. This was done

by Ellis and is used by him. In our clinic we use Volkmann three tine retractors or retraction by forceps only.

5. *The Drainage Tube* (Plate VII).—After the bone work of the mastoidectomy is complete a drainage tube is put in place. For this is used a piece of rubber catheter (F 16). A long fenestrum is cut in the middle and one small perforation just distally toward each end and on the opposite side of the tube. (Plate VIII.) The ends of this tube extend about three-eighths of an inch beyond the incision after the closure is complete. The long fenestrum is turned so as to look toward the antrum. After completion of the bone work the entire field, bony and soft tissue, is thoroughly sponged with mercurochrome special solution.* This technic presupposes a complete exenteration of the cellular mastoid. In our experience an antrotomy or a partial mastoidectomy is unsurgical under the conditions encountered in the average mastoid coming to operation. In case incomplete bone work is done the only course to pursue is the open method with a comparatively long period of packing and drainage.

6. *The Periosteal Closure* (Plate IX).—The periosteal incision is now closed with catgut, either interrupted or preferably continuous. This line of closure, as already noted, is well anterior to the skin closure. Also, if the flap has been properly planned, it is well posterior and beyond the bony excavation except at its very ends, where the skin incision and the periosteal incision unite and where the tube finds exit. The sponging with the mercurochrome special is repeated after this periosteal suture is complete, treating both skin surfaces and the edges of the skin incision.

7. *The Cutaneous Closure* (Plate X).—The skin is now closed with interrupted skin sutures, and, if so desired, with Michel clips. The tube is trimmed as noted above, so as to project about three-eighths or an inch at each end.

8. *Postoperative Care*.—The day following the operation, irrigation with alcohol acetone mercurochrome solution is made through the drainage tube, with a medicine dropper, about a

*The prescription of the mercurochrome special solution is: Mercurochrome, 6; alcohol, 95 per cent, 145; acetone, 45; aqua dist. qs. ad., 500.

dram of mercurochrome solution being used. This procedure is repeated daily until the tube is withdrawn. The sutures are removed on the fifth day. Usually by the next dressing no evidence of discharge is present. As soon as this is true, the upper end of the tube is cut off close to the skin and the tube is drawn downward within the wound until its free upper end rests about over the antrum.

The excessive lower portion thus exposed is only partially removed so as to prevent the tube from slipping upward into the excavation. It may be guarded with a safety pin or a suture. In another day or two, when it is evident that the discharge through the external auditory canal has ceased and there is no further discharge through the tube, it is completely withdrawn. Usually this is accomplished by the sixth or seventh dressing. Within forty-eight hours more the wound is practically healed so that only the slightest dressing is necessary after the first week. Ellis usually removes the tube completely, after cutting off its exposed upper extremity, at one step on the sixth or seventh day. After he removes the tube he still flushes the cavity with the mercurochrome special through the upper or lower skin opening, for a day or two longer.

9. *Advantages.*—The objection may be raised against the flap that the technic is tedious. That it is tedious cannot be denied, but the few extra minutes required to make the flap are repaid many times over because:

(a) The flap always heals by primary union. This is obviously a blood clot method, with a certain safety valve advantage. The failure of the clot to organize with the resultant unhappy breaking down and opening up of the wound is avoided. The absence of the suture line over the blood clot doubtless minimizes the possibility of infection from the skin.

(b) Convalescence is so definitely shortened.

(c) Since the wound is completely closed, except for the drainage tube, the dressings may be well and safely done by an assistant.

(d) Postoperative dressings are practically painless.

(e) The line of cutaneous incision insures a minimum of visible scarring. The scar is of the linear type, and even that to great extent within the hair line.

(f) The normal contour of the mastoid region is retained. The unbroken periosteal surface presented over the bony excavation encourages prompt healing within this cavity, and doubtless makes for bone re-formation. Data on this point is scanty, only two cases having been dissected, several months following the operation.

The few failures in the hands of a dozen or more surgeons who have tried this technic may be traced to technical errors in certain important details. When any of you men try this operation and have suggestions, questions or possibly poor success, Dr Ellis or myself would be glad to hear from you. It is our belief that if the procedure is carefully performed success will result. On the other hand, it is quite probable that from the experience of many surgeons certain refinements may be developed.

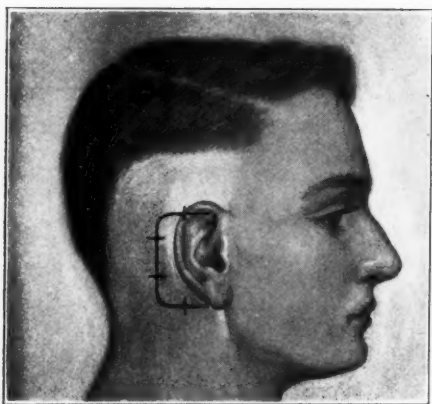


Plate 1. Area of shaving and skin preparation.

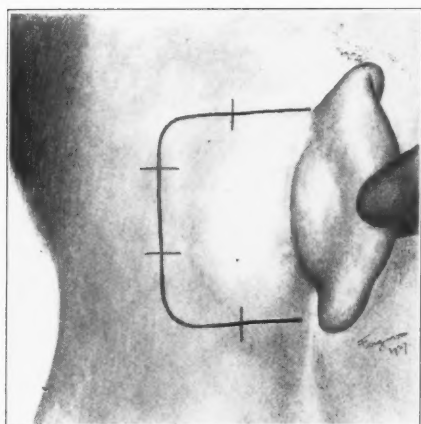


Plate 2. Mapping of skin incision.

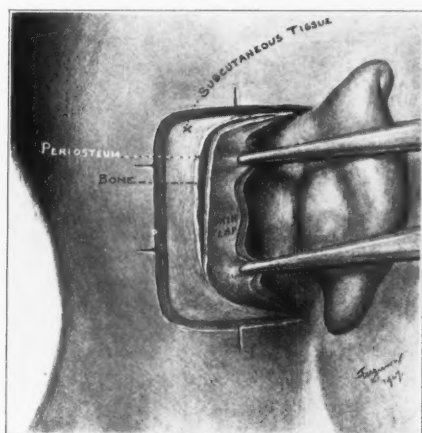


Plate 3. Skin incision, skin flap and beginning of periosteal incision.



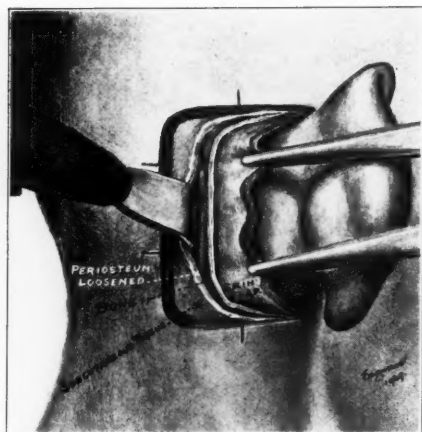


Plate 4. Completion of periosteal incision and elevation of posterior portion.

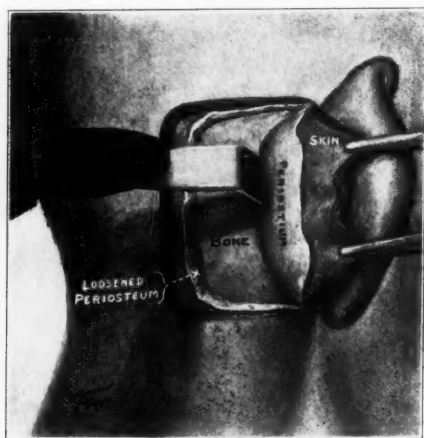


Plate 5. Elevation of anterior periosteal apron and skin flap with bone exposure.

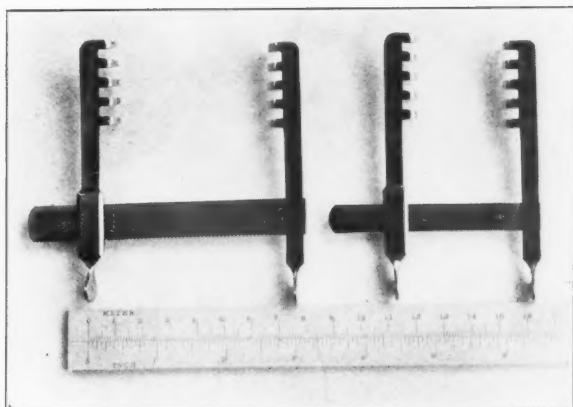


Plate 6. Andrews selfretaining retractor with the slide arm modified by Ellis to measure $3\frac{3}{4}$ inches.

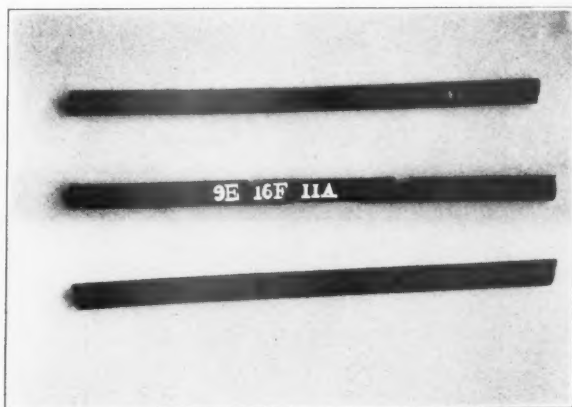


Plate 7. Typical drainage tubes, rubber catheter (F16) showing three fenestra.



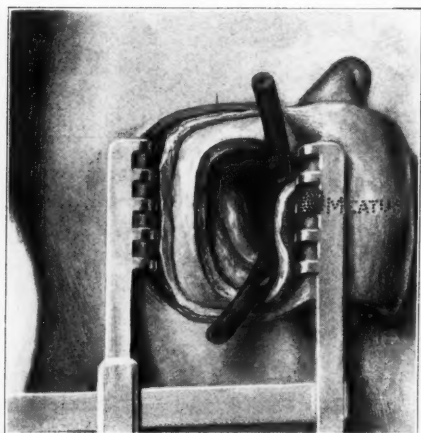


Plate 8. Bonework complete, showing retractors in use, and tube laid in position.

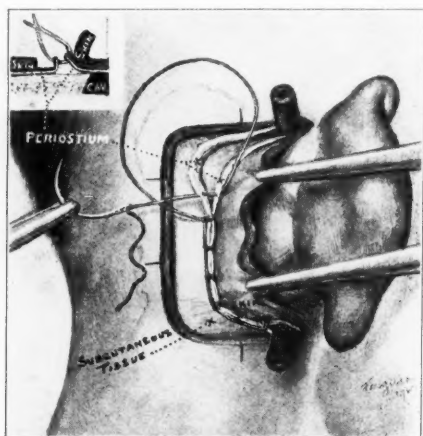


Plate 9. Periosteal closure, tube in place. Inset shows catgut suture through free edges of periosteum.

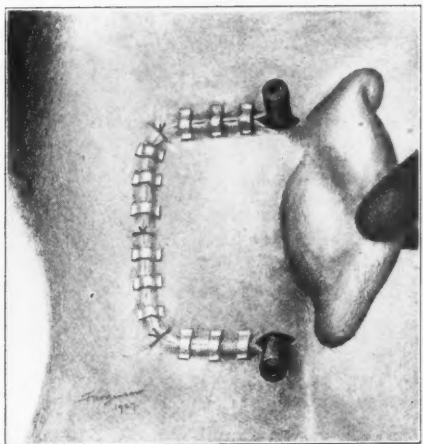


Plate 10. Cutaneous incision closed. Tube in place.



Plate 11. Patient on sixth day.



Plate 12. Same patient on twenty-first day.



Plate 13. Patient on twelfth day, showing unusually long flap made necessary by very extensive cellular distribution.

XLIII.

PERFORATIONS OF THE ESOPHAGUS.*

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This paper deals with a break in the continuity of the esophagus due to pointed foreign bodies, instruments or local morbid processes, capable of sudden penetration.

Of sharp foreign bodies the fish bones, the chicken bones, splinters of beef bone, and pins of various types, are the most frequent offenders; though any sharp object capable of penetration must be considered. Thus a sharp piece of metal, which was thought to come from a dental filling, was responsible for the perforation in one of Brown's¹ cases.

Instruments would include esophagoscopes, gastroscopes, bougies, stomach tubes, probangs, coin catchers and various types of grasping forceps.

Under morbid processes, the most frequent cause of perforation of the esophagus is carcinoma, though other conditions are to be considered. Thus Freeman reports a case of tracheoesophageal fistula which he believed was due to syphilis, and Frey¹⁰ reports a case of perforation of a traction diverticulum into the right bronchus, causing a lung abscess. It is the weakening and gradual thinning of the esophageal wall that causes an opening in it in these cases. The process of perforation of the esophagus in this type of case is a rather slow one; therefore, a perforating carcinoma of the esophagus, for instance, is not acute unless the carcinoma has penetrated a neighboring structure of importance, as, for instance, the aorta.

The recognition of the presence of an esophageal perforation and an understanding of the train of symptoms which it may induce are of value. At the present time perforations of the

*Read before the American Bronchoscopic Society, Atlantic City, May, 1927.

cervical esophagus can be handled in time and adequately, so that the mortality from this type of case in the larger clinics is comparatively small and is no doubt decreasing. Perforations of the thoracic esophagus, however, are not considered to be surgically curable at the present time. This is for two reasons. First, the perforation is usually not recognized at once, and secondly, the thoracic surgeon is not consulted at all, or if he is consulted he is called in when the patient will not survive any type of surgery. We should, therefore, strive for early recognition and immediate surgery for these thoracic cases.

This paper does not include perforations of the esophagus from lesions primarily outside of the esophagus, such as the transpleural esophageal fistula of Ballin¹ and Saltzstein.

Perforations of the esophagus may be divided according to location into cervical and thoracic. In the cervical region the penetration of a foreign body will cause local reaction, which develops as a cellulitis, with or without abscess formation. With this reaction there may or may not be subcutaneous emphysema. In a great majority of cases there is some emphysema present. In many of these cases there is abscess formation which requires surgical drainage. Perforation of the esophagus in this region by an instrument, such as an esophagoscope or a bougie, usually communicates with the pleural cavity. In such cases there is an immediate production of a pneumothorax, and within twenty-four hours there is evidence of a beginning pyopneumothorax. Where the perforation is irregular and the connective tissue planes are opened, subcutaneous emphysema may complicate the picture.

The following cases illustrate perforations of the cervical esophagus:

Case 1.—C. B., age 12 months, was admitted to a hospital June 27, 1925, with a penny lodged in the esophagus for the past fifteen days. An attempt at removal of the intruder, which was located just below the cricopharyngeus, was made with a 5 mm. bronchoscope by the laryngologist in attendance. The foreign body was not seen by the examiner, but during his attempt at introduction of the tube a false passage was made through the right side of the upper esophagus. The child was seen by me fifteen hours later. She was in extreme shock. The

right lung was completely collapsed and the mediastinum was pushed well over to the opposite side. The foreign body was removed but the child died soon after. A sudden pneumothorax had been caused by perforation into the pleural cavity, causing death within sixteen hours. (See Fig. 1.)

Case 2.—H. D. P., age 65, was admitted to the service of Dr. Yankauer, at the Mount Sinai Hospital, September 14, 1926. He had been suffering with difficulty in swallowing for the past month. There was positive X-ray evidence of a carcinoma of the esophagus in its thoracic portion. In attempting to perform esophagoscopy for diagnosis, the medium sized Mosher esophagoscope forced a false passage along the line of cleavage between the esophageal and left tracheal walls for a distance of 5 cm. As the esophagoscope was passed downward I was struck with the delicate connective tissue fibers which I saw. The esophagoscope was withdrawn and passed into the esophageal lumen. An irregular cauliflower like mass was demonstrated 30 cm. from the upper tooth mark. Immediately after withdrawal of the esophagoscope crepitation could be felt beneath the skin of the left side of the neck. The patient had some difficulty in breathing and some pain. There was no noticeable increase in the respiratory rate. The heart was not displaced. Six hours after esophagoscopy the temperature was 100.2°.

I was strongly in favor of external operation, but Dr. Yankauer and Dr. A. V. Moschowitz advised against it. The next day the patient could not swallow because of deep seated emphysema and local reaction, so that a gastrostomy was performed. At this time his temperature was 103°. Two days later crepitation was still present, and there was marked difficulty in swallowing. On the fifth day the temperature began to decline, although swallowing was still very difficult. On the seventh day after the mishap, all evidence of trouble in the neck had disappeared.

The above two cases demonstrate that the introitus of the esophagus is a real danger zone and should be approached with extreme care.

Case 3.—J. P., a boy of 8, swallowed a very small safety pin, such as is attached to neckties. He was seen on November

9, 1925, a few hours after, at the Beth Israel Hospital. Search with the upper esophageal speculum was unsuccessful in locating the pin, although the roentgen film placed it definitely in the upper esophagus. At this time there was noted a small laceration in the posterior hypopharyngeal wall, and the posterior esophageal wall at the cricopharyngeus bulged forward noticeably. This was probably due to air behind the esophagus. At fluoroscopy, the pin was removed in a few seconds. Fluoroscopy demonstrated the pin lying in a horizontal plane, higher up in the hypopharynx, whereas, before, it appeared in a vertical plane with the point downward. Twenty-four hours later there developed a temperature of 100.8° and emphysema of the tissues of the left side of the neck. There was some pain on pressure. Forty-eight hours later, the temperature had risen to 103.2° , and there was less emphysema but considerably more pain. Operation was performed by Dr. Seff, who drained an abscess cavity, deeply placed, which contained about one-half ounce of pus. The sheath of the jugular was edematous and slightly necrosed and contained pus. Air could be seen escaping with the respiratory movements. The venous flow in the jugular vein was unimpeded.

On the morning after operation the child was more comfortable. That evening, however, he had a chill and a sudden rise of temperature to 106° . The internal jugular was, therefore, ligated and resected. He was more comfortable, but on the fifth day following the mishap (day after ligation) he developed signs of congestion of the right upper lobe of his lung. From that time on he showed signs of a septicemia with changing manifestations in his chest. He finally died, forty-eight days after admission, having had several transfusions and infusions of mercurochrome, 1 per cent.

Autopsy revealed free seropurulent fluid in the right chest cavity. There were multiple abscess cavities in the middle lobe of the right lung and a large hemorrhagic infarct in the lower lobe of the same side. The left upper lobe and the upper portion of the lower lobe contained many bronchopneumonic patches with small abscesses. The lower lobe contained a large abscess cavity, filled with foul smelling pus. There was no gross evidence of perforation of the esophagus.

Comment: This case demonstrates the serious sequelæ of a minute esophageal perforation, which at the time it occurred was considered perfectly benign. Here the jugular vein was the site of a septic thrombophlebitis, which later caused multiple infections of the lung, due to emboli.

Case 4.—C. B., age 58, was seen at the Jewish Hospital April 18, 1926. She gave a history of having swallowed a chicken bone the day before. The left pyriform fossa was seen to be markedly edematous, as was also the left arytenoid. A thin chicken bone was found transfixing the posterior surface of the left arytenoid and penetrating the lateral wall of the left pyriform fossa. The next day the edema had increased in the region of the lodgment of the foreign body. Three days later the local pain and tenderness had increased, the edema had increased, and in addition there was a spasm of the left sternomastoid muscle. The temperature was now 103.2°. There was at no time subcutaneous emphysema. This patient was seen by Dr. Imperatori, who agreed that operation was indicated. A collection of whitish, foul smelling pus was evacuated from behind the lateral lobe of the thyroid gland. On the day after operation, the edema was almost entirely gone. At this time it was discovered that the patient was a diabetic. Twelve days after admission the patient's temperature had fallen to normal and she was swallowing without difficulty. She was now being kept in the hospital for her diabetes. After a period of twenty-one days, during which there was no evidence of trouble in her neck, she developed chills and a high temperature, with respiratory distress. She seemed to have a sense of oppression beneath the sternum. She became profoundly ill and died of what was thought to be a septicemia, seven days later. Dr. Lilienthal saw this patient forty-eight hours before she died and suspected a septic thrombosis of one of the thoracic veins.

Comment: This case presents an unexpected sequel in the form of a septicemia and death after a period of apparent recovery which lasted twenty-one days.

Case 5.—P. L., a girl of 15, was seen July 4, 1926, four days after she had swallowed a very small fish bone. She had suffered with increasing difficulty in swallowing and now complained of considerable pain in the right side of the neck.

Exquisite tenderness was elicited on pressure. She had a temperature of 104° .

With an upper esophageal speculum a bulging mass was encountered in the lower portion of the right pyriform fossa. This was incised and considerable foul smelling pus was evacuated. No bone was encountered. The next day the temperature had not subsided and the same clinical picture was present. An external operation was, therefore, performed. During the dissection in the region of the carotid sheath and behind the lateral lobe of the thyroid a large quantity of pus was emptied into the pharynx. Only a very little pus was encountered through the external wound. The bone was not seen. On the same day of the operation the patient complained of severe pain in the lower esophageal region, which persisted for twenty-four hours. I have seen the same type of referred pain in a case with a chicken bone in the upper esophagus. The wound gradually healed, but at the end of the two weeks a small sinus still persisted. More than one month later a nurse found a very small fragment of bone (one-eighth inch long) protruding from the wound. After this was removed, the wound closed.

Comment: This case demonstrates a symptom of referred pain localized to the region of the lower esophagus in upper esophageal foreign bodies.

Case 6.—D. O., age 58, was admitted to Dr. Yankauer's service September 12, 1926, having swallowed a fish bone three days before. Her first sensation was pain in the back and left side of the neck. She complained of pain on swallowing and pain on coughing. This pain she located directly beneath the sternum. At upper esophagoscopy, the left pyriform fossa was found filled with pus, which upon aspiration disclosed a thick bone about $1\frac{1}{2}$ inches long, lying free in this region. Pus was seen to be welling up from the cricopharyngeus area. She was kept under observation because pus kept coming out of the opening in this region. Of interest is the fact that this patient had no clinical evidence of abscess formation and that the presence of pus was not suspected until she was examined with the laryngeal mirror. She left the hospital six days after esophagoscopy, refusing to stay any longer, because she felt so

well. At this time she still had a small amount of pus in her left pyriform fossa.

Case 7.—M. H., age 45, was seen at the Jewish Hospital September 25, 1926. She was suffering from pain and difficulty in swallowing because of a fish bone she had swallowed three days previously. A laryngologist had seen her immediately after the mishap and had advised her to wait three days to see if anything would happen. When seen, she had slight emphysema of the right side of the neck, considerable pain and tenderness and some swelling in the central portion of the neck in the region of the sternomastoid. Her temperature was 101.6° . Removal was accomplished with an upper esophageal speculum. At this time the right pyriform fossa in its lowermost portion was swollen and edematous. No pus was encountered. The temperature subsided within the next two days, and the patient made an uneventful recovery.

Comment: The above two cases demonstrate that Nature may so wall off infection following the penetration of sharp foreign bodies in the cervical region as to insure recovery after endoscopic removal.

Case 8.—R. B., age 48, was seen at the Jewish Hospital November 13, 1926, because she had swallowed a fish bone a few days before. Her temperature was 102° , and she was complaining of pain and difficulty in swallowing. She presented the same type of external picture as Case 7, yet at esophagoscopy pus was found in the left pyriform fossa, and the bone was removed from the left upper esophageal wall, where it was impacted. Because of a persistence of symptoms, and advancing edema of the left external laryngeal wall, external operation was performed. A very small amount of foul smelling pus was evacuated from behind the lateral lobe of the thyroid. The patient made an uneventful recovery and was discharged ten days after admission.

In the thoracic portion of the esophagus the patient's chances of recovery after perforation are not so great as in the cervical portion. The greatest obstacle to the proper surgical care of such an emergency is the comparative inaccessibility of this region. The reaction of the adjacent structures which may be involved in the perforation is of great importance in this respect.

Roughly speaking, the esophagus is in contact with the following structures in its thoracic portion: The trachea, left bronchus, the vertebral column, the aorta, pleura and pericardium. On the esophageal wall we have the periesophageal venous plexus, whose superior branches drain into the thyroid veins, while the inferior branches go to the azygos veins.

The following cases illustrate perforation of the thoracic esophagus:

Case 9.—J. S., age 39, was seen on the service of Dr. Yankauer September 7, 1926, because he complained of severe pain in the back and inability to swallow. He also had pain on coughing, pain on deep breathing and he expectorated a yellowish sputum which was foul smelling. He had swallowed a chicken bone seven days before. Esophagoscopy revealed a large area of redness and swelling posteriorly, 2 inches below the cricopharyngeus. The pressure of the lip of the Mosher esophagoscope against this swelling caused a flow of pus, which was removed with the suction apparatus. Because of the presence of the pus, it was thought inadvisable to search for the bone. A few hours later the temperature rose to 103.2°. The next day he complained of pain in the right side of the neck which was deep seated and aggravated by swallowing. Subcutaneous emphysema was present and was more pronounced in the episternal region and extending along the right side of the neck. Evidently the foreign body, which had acted as a cork in the perforation, had been disturbed from its position and air was being forced through the tissues. Two days later the pain in the throat was much less and the patient could swallow. The emphysema was almost entirely gone. Six days later, temperature was normal but he was expectorating a thick mucopus. On the ninth day the patient had enjoyed his third day of normal temperature, and esophagoscopy for removal of the foreign body was performed. An irregularly triangular bone, 1½ inch long, was found lying horizontally in the upper esophagus. Fourteen days after admission, esophagoscopy was performed and granulations were noted in the region of the previously encountered wound. The patient was discharged.

Comment: Here we have an example of Nature's success in localizing the suppurative process which the perforation induced. The disturbance of this area by the esophagoscope

and the moving of the foreign body caused considerable local reaction. Here again we have a case with subcutaneous emphysema and fairly high temperature, which recovered spontaneously.

The next two cases are fatalities, due to bouginage, in lye strictures of the esophagus:

Case 10.—No. 2166, age 11, was seen on the service of Dr. Yankauer, October 26, 1926. He complained of a progressive difficulty in swallowing for the past three months. Three months ago he was given a drink of what somebody told him was beer and which proved to be a solution of lye. He had lost about twenty-five pounds. The X-ray report stated that he had an obstruction almost complete on a level with the sternoclavicular junction. The esophagus above this point was moderately dilated. Opinion was benign stricture. Esophagoscopy revealed a stenosis in the cricopharyngeus area of about 1 mm. diameter. At this time I was able to pass a No. 16 French bougie through the stricture. As a result, he could swallow semisolid and liquid foods without difficulty. A few days later I was able to pass a No. 21 French bougie through the strictured area. But in passing the bougie, I permitted it to drop all the way down into what I thought was his stomach. Immediately upon withdrawal of the bougie the boy complained of severe pain in the left lower chest, which shifted in a few minutes to the right side. He now had severe pain in the lower back and abdomen. The breath sounds were markedly diminished on the right side from the lower border of the scapula to the base. There was a slight displacement of the heart to the left. These signs and the severe pain indicated a perforation of the esophagus, probably in its lower portion. The next day there were definite signs of a pneumothorax of the right side. Forty-eight hours later a needle was introduced into the fourth interspace anteriorly, and air was permitted to escape under water. The respirations became more labored, the temperature rose to 105° , and the child died three days after perforation.

Autopsy revealed: The right thoracic cavity contained about 1,000 cc. of yellowish green purulent material, which had pushed over and collapsed the lung medially to about one-fourth its original size. The entire mediastinum was pushed

to the left side. The pleura was covered in its entirety with a thin layer of fibrin. Exploration of the right thoracic cavity after the fluid was emptied disclosed a small oval perforation of the esophagus measuring about 4 mm. in diameter and located posteriorly about 2 cm. above the diaphragm. Slight pressure upon the stomach caused the gastric content to flow into the right thoracic cavity through the perforation.

On opening the stomach and esophagus in situ, there is a circular area of scarring found at the level of the first dorsal vertebra and extending about 3 cm. downward. About 6 cm. below this there is the beginning of a second zone of constriction which extends downward about 4 cm. At the beginning of the second zone of constriction posteriorly, and slightly to the left, there is a small perforation about $\frac{1}{2}$ cm. in diameter. A probe inserted in the constriction passes readily downward and to the right about 5 cm., and makes its exit at the perforation described in the right thoracic cavity above the diaphragm. Microscopic examination of the esophagus showed edema and thickening with small areas of ulceration and of inflammatory infiltration.

Comment: In this case, unfortunately, I permitted myself to deviate from the rule of insisting upon the use of a previously swallowed thread as a guide in dilatation of the stricture. In addition, I did not have the X-ray film in the operating room at the time of the esophagoscopy and the use of the bougie. A study of the film at the time of esophagoscopy is very important. In this case there was evidence in the roentgenogram of a lower stricture. And the last offense and most serious one was the dropping of the bougie down the esophagus to see if the patient felt it in his stomach. This procedure should be condemned as a dangerous practice.

It should always be borne in mind that in lye poisoning cases there are usually multiple strictures. Therefore each stricture should be considered separately. My experience, both with successes and failures in this work, has made me a firm believer in the absolute necessity of having a previously swallowed thread as a guide. In my hands, a bougie used in any type of stricture without a string guide is as dangerous and unjustifiable as the much deprecated blind bouginage. To me it has come to mean blind bouginage, even though we may be

able to see where the bougie enters the upper limit of a stenosed lumen.

Case 11.—No. 2167, age 27, was admitted to the service of Dr. Moses of Kings County Hospital, January 11, 1927. He had just swallowed a strong solution of lye in an attempt at suicide. As a result of a previous encephalitis, he was suffering with a Parkinsonian syndrome. In addition, he had developed a suicidal tendency. X-ray study indicated a marked stenosis of the upper esophagus. Dilatation was carried out with the Plummer-Vinson bougies over a previously swallowed thread, on three occasions. At no time during dilatation was there any reaction on the part of the patient to make us feel that he had been injured. The last dilatation was carried out to a No. 30 French measurement. Four days after this the patient complained of pain in his chest and began to cough. Thereafter he expectorated purulent sputum in large quantity. X-ray showed an incomplete consolidation of the lower portion of the left upper lobe. Bronchoscopy confirmed the presence of a left upper lobe suppuration. At this time a barium study was made of the esophagus, and Dr. Rendich noted that a small amount of the opaque mixture had trickled into the superior mediastinum, suggesting a small perforation. Although he could swallow liquids and soft foods fairly well, the patient gradually succumbed to the effects of his lung suppuration.

It is of interest to note that four days after the last dilatation the patient experienced pain in his left chest and began to cough. From this time on there was present the evidence of lung suppuration. Whether the esophagopulmonary communication noted above was a direct result of the dilatation or was the result of a spontaneous tear during exertion is not clear. The greater likelihood, however, is that the dilatation was responsible for this complication. This case again proves the necessity for the utmost care in endoesophageal manipulations.

A case in point is that reported by Eguen,⁹ where a boy of four, with a lye stricture of two years' duration, suffered a spontaneous perforation at the posterior esophageal wall, at the level of the aortic arch. Barium was seen to flow into the right upper lobe of the lung. There was some reaction, but the perforation healed in three weeks.

Friedenwald¹¹ and Morrison report three cases of perforations of the esophagus due to instruments, and state that "the dangers involved do not appear to have been sufficiently emphasized."

In the first case, a bougie was threaded through the esophagus with the aid of a gastrostomy wound. With the second bougie, something was felt to give. In the second case esophagoscopy followed several radium exposures. Immediately after esophagoscopy the patient complained of severe substernal pain with marked dyspnea and cough. In the third case there was severe pain immediately after an attempt to dilate the esophagus. A perforation was found into the right lower lobe bronchus, which had caused a bronchopneumonia and death.

Case 12.—This last case represents the perforation of an unsuspected carcinoma of the esophagus through the aorta. M. H., age not given, came to the Kings County Hospital, January 15, 1921, because she had coughed up some blood in the morning. She had caught cold six months ago and ever since then had been weak. She had been coughing for the past three weeks and had lost considerable weight. She also complained of difficulty in breathing. Except for being somewhat emaciated and having a rapid pulse there were no significant physical signs. On the day after admission she expectorated about one ounce of blood. Forty hours after admission she vomited about two ounces of bloody mucus and died rather suddenly within the next few minutes.

Autopsy revealed: The esophagus in its middle portion presented a large and apparently malignant growth, which filled the entire posterior mediastinum and involved the ascending arch of the aorta and the trachea. It was about five inches in width and entirely surrounded the esophagus. Upon dissection a sinus communicating with the aorta was found. The esophagus was filled with blood. The stomach and the remainder of the gastrointestinal tract contained free and clotted blood. Cause of death, internal hemorrhage due to rupture of aorta into the esophagus.

The unusual feature of this case was that we were dealing with a squamous cell carcinoma of the esophagus which at no time caused difficulty in swallowing.

A review of the more recent literature is replete with many interesting and unusual cases of esophageal perforation.

Miller¹⁸ reported the case of a girl of ten, who during the night was seized with pain in the epigastrium and nausea. She was in profound shock and markedly pale. The type of breathing suggested a foreign body in the bronchus. She died within forty-eight hours. Autopsy revealed a narrow slit, about 1 centimeter long, on the left side of the esophagus, 5 centimeters above the cardiac opening of the stomach. She had a left pyopneumothorax. No foreign body was found.

Among perforations not due to foreign body the case of Christie⁶ is of interest. A man of sixty was operated upon for what was thought to be a ruptured gastric ulcer. He died three hours later. Autopsy revealed that a simple ulcer of the lower esophagus had perforated into the thoracic cavity two inches above the diaphragm.

Coates⁷ and Goepp recorded the case of a lady who, after swallowing what was probably a sharp chicken bone, developed a variety of septic phenomena, such as urticaria, pleurisy, nephritis, polyarthritides, severe secondary anemia, and ultimately recovered. Linn¹⁶ reported a fatal case of perforation of the aorta by a chicken bone. Similar accidents have been recorded by Beal² and Doria.⁸ In Doria's case the patient died suddenly of hemorrhage, and the cause of death was thought to be pulmonary tuberculosis. Autopsy, however, revealed the presence of a bone 2½ cm. long, which had caused a perforation of the aorta after the esophageal wall had ulcerated.

Iglauer¹³ and Ransohoff brought forth a new diagnostic sign in the roentgen examination of foreign body impacted in the posterior esophageal or pharyngeal wall, and reported an interesting case.

Jackson¹⁴ mentions five instances of esophageal abscess, and cites a case in which there was septic mediastinitis with cellulitis of the neck. The abscess was evacuated and the patient recovered. No bone was found.

Campbell's⁵ case, in which two anterior sacculations of the upper esophagus were found, after strictures due to the ingestion of lysol, and three years later, hydrochloric acid, is unusual. Forty-two hours after esophagoscopy the patient died. It was revealed at autopsy that the lower of the

two sacculations had perforated and caused a suppurative pericarditis and empyema.

Perhaps the most interesting case of its kind is the experience of Maclay.¹⁷ Endoscopic search for a fish bone revealed a granulating area about $1\frac{1}{2}$ inches below the cricopharyngeus. Cellulitis of the neck followed, and later pressure upon the right side of the neck caused pus to enter the throat. External operation one week after onset disclosed a large collection of pus, which was drained. One month after the onset an abscess over the right second intercostal space was incised, and it was found to communicate with the superior mediastinum. This abscess cavity communicated with the original abscess in the deep tissues of the neck. The wound kept open for six weeks and finally healed. The author believed he had perforated the upper esophagus with his tube.

A majority of the bones swallowed by adults in my series of bone cases have occurred in individuals who wear a set of false teeth in their upper jaw. This unhappily subjects the individual to the danger of ingestion of sharp particles of food because he is deprived of the exposed mucous membrane of the hard palate, which ordinarily warns one of the presence in his mouth of foreign or sharp substances. Guthrie¹² also stresses the point that a majority of his patients were edentulous. Guthrie and Holland report a series of eleven cases of meat and fish bones in the esophagus. Holland mentions the value of X-ray and fluoroscopy in these cases. In one of their cases inflammatory reaction was marked, a definite abscess discharging pus being present. There was also much external swelling of the neck, which gradually subsided after removal of the bone.

Berry³ had an interesting experience in which a lady injured her upper esophagus in dislodging a foreign body. She developed emphysema and abscess; sepsis supervened, and she finally died, nine days after injury.

A word is in order concerning the indications for external operation in hypopharyngeal and upper esophageal perforations. The most important and first symptom in this type of accident is pain. This pain may be immediately followed by emphysema and swelling, either alone or in combination. Fever soon supervenes if infection is present. The experiences cited

above would indicate that external operation may be deferred, even in the presence of severe local reaction and a systemic reaction denoting absorption, fever. This does not conform with the view expressed by H. Killian¹⁵ and Schlemmer.¹⁹ In a review of a large series of cases, Killian makes the presence of emphysema in upper esophageal perforations an absolute indication for operation. Schlemmer, in reviewing ten years of experience, takes the same stand as Killian. Von Eicken,²⁰ however, has come to the conclusion that emphysema is not necessarily an indication for external operation. He feels that it is better to wait and watch. He cites eight cases in which the local process subsided spontaneously. These authors use the term "esophagotomy." In the cases which I have operated upon, esophagotomy was not performed. An anterior sternomastoid incision was made; then by dissection through the lines of cleavage the localized suppurative process was encountered in the deep cervical fascia, usually situated directly behind the lateral lobe of the thyroid, and outside of the esophageal lumen. In these cases it is felt that the suppuration has localized itself entirely outside of the esophagus and that the perforation that initiated the process has usually healed before the time of operation. In these cases a direct communication with the esophageal lumen was not established as a result of the operation. The term esophagotomy does not fit the above procedure, which is really a dissection and drainage of the deep tissues of the neck.

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30 EAST 40TH STREET.

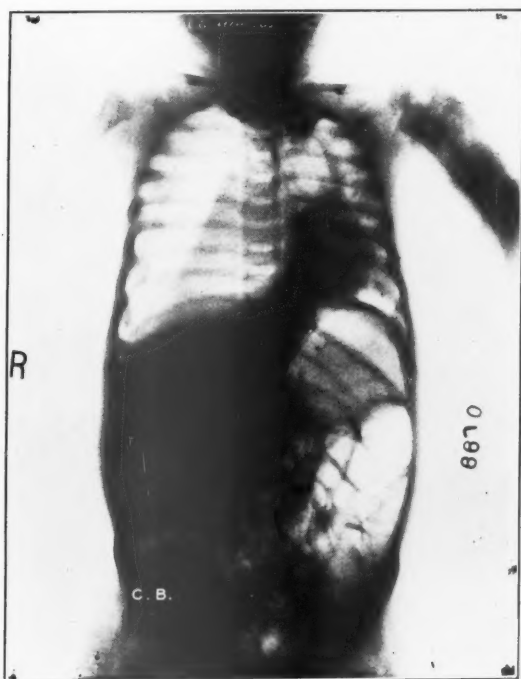


Fig. 1. Case 1.—Showing pneumothorax with displacement of mediastinum due to perforation of esophagus.



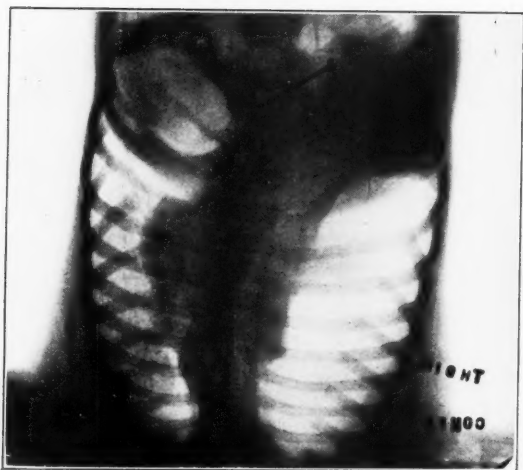


Fig. 2. Similar to Case 1, in which the foreign body was pushed into the stomach at the same time the esophagus was perforated.

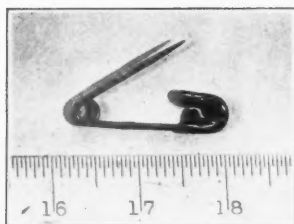


Fig. 3. Case 3.—Small safety pin successfully removed, but minute perforation caused disastrous sequelae.



Fig. 4. Case 4.—Chicken bone removed from infected area in left pyriform fossa. Followed by unexpected sequel and death.





Fig. 5. Case 5.—Small fragment of fish bone perforated, caused infection necessitating external operation. Bone delivered itself some time later through fistula in neck.



Fig. 6. Case 6.—Fish bone found in a pool of pus in left pyriform fossa; no symptom of infection present.



Fig. 7. Case 8.—Penetrating fish bone which caused infection necessitating external operation.



Fig. 8. Case 9.—Chicken bone impacted in thoracic esophagus; caused infection which subsided after removal.



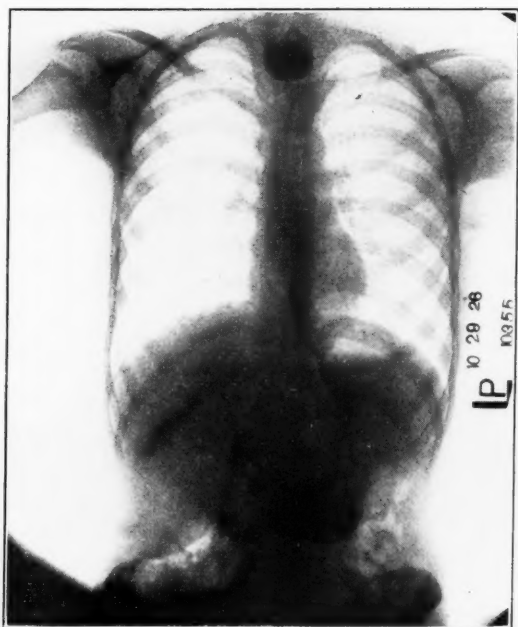


Fig. 9. Case 10.—Lye stricture of both upper and lower ends of the esophagus. In the procedure of bouginage the lower stricture was overlooked and a tunnel perforation of the esophagus resulted.



Fig. 10. Case 11.—Lye stricture of the upper esophagus, which was followed by an esophagopulmonary fistula several days after the last passage of a bougie over a previously swallowed thread.





Fig. 11. Case 11.—Same patient. Showing lung abscess in left upper lobe and the presence of some barium mixture in the left lung which entered from the esophagus.

XLIV.

POSTOPERATIVE ATELECTASIS—BRONCHOSCOPIC OBSERVATIONS ON MASSIVE COLLAPSE OF THE LUNGS.*

BY GABRIEL TUCKER, M. D.,

PHILADELPHIA.

The first bronchoscopic examination of a patient with postoperative massive collapse of the lung was made January 14, 1925, on a boy 13 years of age. The condition followed an appendectomy, under ether anesthesia, on the service of Dr. Walter Estell Lee at the Germantown Hospital, Philadelphia. The details of bronchoscopic studies in this case were reported at a meeting of the College of Physicians of Philadelphia by Lee and Tucker, and recorded in the 1925 Proceedings of the College. From that time to the present the available literature shows only four cases of postoperative massive collapse reported in which bronchoscopic study was made.

The second case observed bronchoscopically occurred in a patient, 6 years of age, admitted to the bronchoscopic and surgical services of the Jefferson Hospital, Philadelphia, and at Dr. Jackson's suggestion a preliminary report of this case was made by the author, who had observed the patient bronchoscopically on several occasions. Later the same case was reported in greater detail by Dr. W. P. Hearn of the surgical service and Dr. Louis H. Clerf of the bronchoscopic clinic.

The third case was reported by Dr. Harrington of the Mayo Clinic. The report of the bronchoscopic findings stated that after aspiration of about 300 cc. of fluid from the left lung there was immediate relief from cyanosis and dyspnea. Further detail of the bronchoscopic study was not given.

In the fourth case, report by Dr. Leroy Sante, mention is made of the bronchoscopic aspiration of secretion by Dr. I. Dee Kelly, without full detail of bronchoscopic findings, but it was

*Read before the American Bronchoscopic Society at Atlantic City May 21, 1927.

observed that the atelectatic lung expanded, after turning the patient over onto the uninvolved side and producing cough. No credit, however, was given to the bronchoscopic aspiration of the obstructing secretion from the larger bronchi.

From the observations of the first and second cases reported, Dr. Chevalier Jackson's opinion was that the condition was definitely one of obstructive atelectasis in these cases. In the other two cases reported the secretions evidently were of different character. It would seem of very great value for further study of the mechanism in these cases if definite points could be recorded and reported with sufficient uniformity in each case to be of a statistical value. In order to offer this suggestion to bronchoscopists, and to report the bronchoscopic findings in another case of postoperative atelectasis, I am presenting this subject.

During the period after the first bronchoscopic observations were made a large number of cases of postoperative massive collapse of the lung in which no bronchoscopy was done have been reported. One explanation for this, is that a bronchoscopist is not always available. It is gratifying, however, to note that Harrington states that the bronchoscopy by Vinson saved the patient's life. As bronchoscopists, I think, we all feel that, where there is no contraindication from the surgical condition, in these cases, it is very important to remove bronchoscopically the obstructing material in the atelectatic portion of the lung.

The first bronchoscopic observation in the first case was made 48 hours after the onset of the collapse; at the time when, according to the X-ray findings, the greatest area of lung was atelectatic. It seems important in reporting the bronchoscopic findings that the time of the examination with reference, first, to the onset of symptoms, and, second, to the degree of atelectasis, should be recorded, because the character of the secretion is likely to change at different stages in the progress of the condition. It is conceivable that the secretions in the bronchus might be fluid at the onset of the collapse, and later become gelatinous, owing to the absorption of the watery content of the retained secretions. This is suggested by the films in one of the cases reported by Leopold, in which the first set of films, made after the onset, showed an opaque lung without

displacement of the mediastinal structures. Films, made 24 hours later, in the same patient, showed displacement toward the affected side. Later in the condition of postoperative atelectasis, we know from observation, that the secretions become liquid in character as the condition clears up. This may possibly be the explanation of the serous nature of the secretion in Harrington's case where it was noted that it poured from the trachea on insertion of the bronchoscope.

I should like to present to the Bronchoscopic Society the findings that were made in the studies of the first case.

First Bronchoscopy.—Preliminary sedative, morphin and atropin, local anesthesia. Abdominal incision protected by wide bands of adhesive encircling the abdomen.

First Examination.—48 hours after the acute onset of collapse. Maximum degree of atelectasis noted in the case was present at the time of bronchoscopy.

Local Conditions.—Mucous membrane of the larynx and trachea slightly reddened, redness being more marked in the lower trachea and right main bronchus. Mucosa of the left bronchus was very slightly reddened.

Secretions.—Small masses of grayish secretion adherent to the trachea; left bronchus free of secretion; right bronchus, a ring of thick tenacious secretion completely surrounded the orifice of the bronchus. Air passed in and out through the central opening in the ring of secretion as it might through the hole in a doughnut. In right upper lobe bronchus, thick secretion adherent to the bronchial wall was found. The right bronchus below the level of the right upper lobe bronchus was completely filled with thick secretion. After aspiration of the secretion a small quantity of air seemed to pass into the middle lobe bronchus. Secretion of the same character was aspirated from the middle lobe bronchus. The lower lobe bronchus with all the main branch bronchi was completely filled with thick secretion, which extended downward as far as aspiration could be carried. The lung had been completely blocked from the level of the upper lobe bronchus downward.

Bronchial obstruction was complete before aspiration in the middle and lower lobes, and partial in the upper lobe on the right side. The trachea and left lung were free of obstruction.

Position and Lumen of the Tracheobronchial Tree.—The lower trachea was deviated toward the right. The carina and right main bronchus were displaced well into the right chest, and seemed less freely movable than normal. The lumen of the trachea and left main bronchus was normal. The lumen of the right bronchus and all its branches seemed normal. There was no evidence of bronchial compression.

Bronchial Movements.—In the atelectatic area, after the aspiration of the secretion, the bronchial movements of inspiratory opening and lengthening and expiratory narrowing and shortening seemed markedly limited. The lumen did not become perceptibly smaller on cough.

Bronchoscopic Diagnosis.—Diffuse bronchitis involving the right lung. Tenacious secretion blocked completely the lower and middle lobe bronchi. Culture of a specimen showed pneumococcus.

The second bronchoscopy was done 72 hours after the first. The X-ray examination at this time showed the right lung clear and the mediastinal structures back in practically the normal position. Locally the mucosa of the larynx, trachea, and right bronchus was found reddened, secretions were now thin, mucopurulent in character, and were found to be coming entirely from the right lung. The position of the tracheobronchial tree was normal; the bronchial movements were normal; there was no evidence of obstruction in the lung that had been atelectatic at the first bronchoscopy.

Bronchoscopic Finding, Second Bronchoscopy.—Diffuse bronchitis involving the right lung, mucopurulent secretion, not obstructive. Right lung functioning well.

This report is not offered as a model but the points mentioned in the report seemed of sufficient value to be included. There may be other points, just as important, which will occur to the bronchoscopist and, of course, should be recorded.

I wish to report the bronchoscopic findings in another case of postoperative atelectasis. A complete report of this case will be made subsequently by Dr. Walter Estell Lee on whose service the case occurred.

A boy, 15 years of age, following an emergency operation for acute suppurative appendicitis, under ether anesthesia, developed a postoperative atelectasis, 24 hours after operation.

Bronchoscopy was done within 36 hours after the onset of the symptoms of collapse. There seemed to be slightly less collapse at the time of the operation than six hours before, from the X-ray examination. (Fig. 1.)

Preliminary sedative, morphin and atropin, with local anesthesia.

Local Conditions.—Laryngeal and tracheal mucosa slightly inflammatory. The right bronchus seemed more inflammatory than the left. The secretion was thick, tenacious and mucopurulent, and considerable quantities were coughed up through the trachea. It was found to be coming from the right bronchus. The left bronchus showed no secretion. The right upper lobe bronchus contained a small amount of secretion and air was passing in and out. The secretion blocked completely the right stem bronchus and all the branch bronchi below this level. The secretion was aspirated and air seemed to enter freely the middle and lower lobes of the lung following the aspiration.

Bronchial obstruction was complete in the middle and lower lobes of the right lung before aspiration.

Position and Lumen of the Tracheobronchial Tree.—There was some displacement toward the right. There was no evidence of bronchial compression after aspiration of secretion. The bronchial movements seemed normal.

Bronchoscopic Diagnosis.—Purulent tracheobronchitis, localized principally in the right, lower and middle lobes. Complete blocking of the right lower and middle lobes.

Following bronchoscopy the patient was able to expectorate secretions much more easily. The X-ray examination showed definite improvement in the aeration in the right lung. (Fig. 2.) Three days after bronchoscopy the atelectasis recurred, but in a few hours free expectoration started, and the further improvement was progressive, the chest was entirely clear of atelectasis at the end of 10 days. (Fig. 3.) The heart was back to its normal position; the secretion changed, becoming thin and mucopurulent, and the lung clear.

In the bronchoscopic examination of these cases there has been no report of evidence of bronchial compression in any case. In fact when the physics of atelectasis is considered it is evident that the atmospheric pressure tends to keep the

bronchus open when the periphery of the lung is collapsed with the pleural cavity closed. This is shown by the fact that all of the mediastinal structures are pushed toward the affected side by the atmospheric pressure. The same pressure maintains the open bronchus where unobstructed, and where the bronchus is obstructed by fluid or semifluid matter, the pressure would be transmitted through the fluid or semifluid substance in the main bronchi and maintain the bronchial lumen. Before the lung can expand it is necessary to remove the obstructing material.

I should like here to note a bronchoscopic observation, the converse of this, that was made in a case, unusual in my experience, in which there occurred a bilateral obstructive emphysema with narrowing of the bronchial lumen. The examination showed that thick tenacious secretions in the main bronchi allowed the air to enter with inspiration but obstructed the expiratory phase that the air was trapped in, producing a high degree of obstructive emphysema. After aspiration of the secretion and relief of the excessive pressure in the periphery of the lung, the bronchial lumen opened more widely. It is a common observation in conditions where there is an excessive pressure outside the lung, as in collapse of the lung due to pneumothorax or hydrothorax that there is a tendency to compression of the bronchial lumen. In this patient the excessive pressure in the periphery of the lung itself narrowed the bronchial lumen and seemed to assist in the trapping of air by the valve-like action of the tenacious secretion. This case will be reported later in detail with further observations on this phenomenon.

CONCLUSIONS.

1. Bronchoscopic observation so far recorded indicate, as Dr. Jackson has observed, that postoperative atelectasis is similar to atelectasis due to foreign body.

2. The mechanism of the production of postoperative atelectasis is somewhat different from that of atelectasis due to foreign body. In foreign body atelectasis the cough mechanism is not usually at fault and the block is only at the point where complete obstruction occurs in the bronchus. In postoperative atelectasis there is interference with the cough mechanism in

the affected portion of the lung and the obstructive secretion, which is in reality a foreign body, extends from the point of complete block into all the smaller bronchi and their branches in the portion of lung distal to this point. From the quotations and comments in the literature on the report of the first case of massive collapse studied bronchoscopically it would seem to be the general impression that the secretion occurred as isolated plugs or crusts throughout the atelectatic area. This was not the case. The bronchoscopic findings indicated that the secretions extended peripherally into all the smaller bronchi, forming as it were, a cast of that portion of the tracheobronchial tree.

3. The bronchi remain dilated in the affected portion of lung in postoperative atelectasis, due to the atmospheric pressure through the lumen of the tracheobronchial tree. The air is absorbed from the alveoli and the portion of the tracheobronchial tree containing the secretion is pushed toward the pleura overlying the atelectatic area of lung.

4. Because of the extension of secretion into the smaller bronchi in postoperative atelectasis the obstructive secretion (foreign body) cannot be completely removed as can the usual foreign body. Recovery is therefore not so rapid as in obstructive atelectasis due to a foreign body where the obstruction can be removed entirely.

5. The impairment of the cough mechanism, with the variations in the character of the secretions at different stages in the progress of postoperative atelectasis offer a possible explanation for the variations in bronchoscopic findings.

6. There are possibly many factors not definitely determined in the mechanism of the production of postoperative atelectasis of the lung. In order to obtain all possible information it is suggested that a detailed report of all bronchoscopic observations be made.

7. Bronchoscopy should be done, or at least the advisability of bronchoscopic operation should be considered, in all cases of so-called postoperative massive collapse of the lungs.

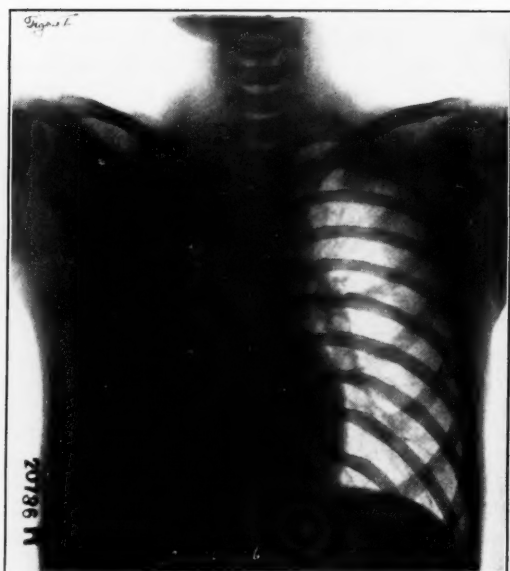


Fig. 1. Roentgenogram showing postoperative atelectasis of the right lung. A patient of Dr. Walter Estelle Lee at the Germantown Hospital, Philadelphia. Film by Dr. Thomas P. Loughery. The heart is displaced well into the right chest and there is evidence of atelectasis of marked degree in the right lower and middle lobes. Film was made just prior to bronchoscopic aspiration of the thick, tenacious secretion from the bronchi.

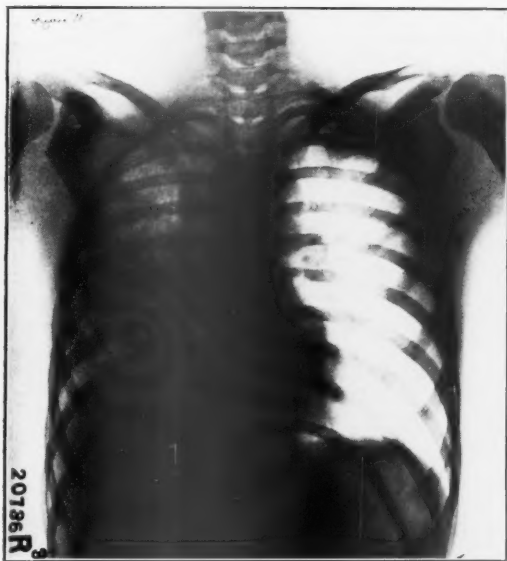


Fig. 2. Roentgenogram of same patient as Fig. 1, immediately following bronchoscopic aspiration. Air is entering all lobes of the right lung. The heart has gone back towards its normal position. The density of the right lung is very markedly decreased. Expectoration of secretion began and atelectasis recurred to some extent but the cough mechanism had become effective and the lung cleared rapidly. The lung was clear at the end of ten days.

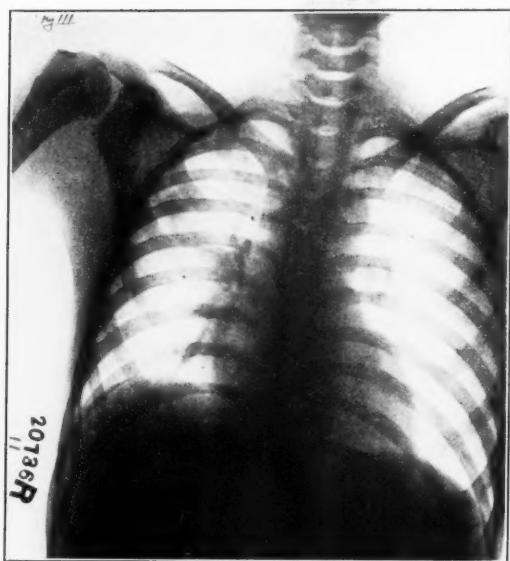


Fig. 3. Roentgenogram of the same patient as Figs. 1 and 2, made two weeks after bronchoscopy showing the lung still clear, there being no return of atelectasis. This case has been reported in detail by Dr. Walter Estelle Lee in the proceedings of the annual meeting of the Pennsylvania Medical Society for 1927. Films by Dr. Thomas P. Loughery.

XLV.

ADENOID DEAFNESS.

By F. PEARCE STURM, M. CH. (ABER.),

LEIGH, LANCASHIRE, ENGLAND.

If there is one fact more than another which otologic experience has impressed upon the writer, it is that chronic progressive deafness begins in childhood. After making every allowance for unavoidable errors in diagnosis and for faulty memories of past disabilities on the part of the patient, it is certain that, at a low estimate, one-third of all cases of acquired deafness in the adult have their origin from preventable causes in childhood or infancy. The accompanying tabulation of 200 adult patients, taken in series from my private practice, is of interest in this connection. The patients complained only of deafness, or deafness and tinnitus. Every one was submitted to a complete otological examination, including the investigation of the upper and lower tone limits, by forks, Galton's whistle and monochord. Cases with active otorrhoea and surgical cases are excluded.

CONDITION DIAGNOSED	No. of Cases
Adenoid deafness with fixation of stapes in both ears.....	16
Adenoid deafness with fixation of stapes in one ear.....	20
Adenoid deafness without definite fixation of stapes.....	28
Neurolabyrinthitis with Ménière's symptom complex.....	30
Neurolabyrinthitis without Ménière's symptom complex.....	8
Otosclerosis (idiopathic degenerative deafness).....	24
Secondary otosclerosis—i. e., otosclerosis with signs of past or present tubotympanic changes.....	32
Acute tubal obstruction—i. e., acute infective tubotympanic catarrh without previous history.....	6
Organic stricture of eustachian tube.....	1
Otomycosis	6
Cholesteatoma	1
Impacted cerumen.....	13
Keratitis obturans.....	3

Synapse deafness (neurasthenia?).....	4
No deafness found (hysteria?).....	1
Undiagnosed.....	7

Thirty-two per cent of these deaf people owed their incurable disability to nasopharyngeal disease in childhood, and must be regarded as cases which were probably preventable. I say probably, because treatment of the adenoid child does not always cure or even prevent deafness. Of these 64 cases, 16 had undergone the removal of both faucial and pharyngeal tonsils before the age of twelve. Of the sixteen cases operated upon, four had a fixed stapes in both ears, two in one ear only, and in ten there were no definite signs of fixation.

Excluding the thirty-two cases of secondary otosclerosis as being only probably and not demonstrably due to preventable causes in childhood, the incidence of adenoid deafness in the series of 200 is 32 per cent. The true figure must be higher, because not only has secondary otosclerosis been ruled out, and some cases of it could legitimately have been included, but active suppurative middle ear disease of any variety or degree has also been excluded. The exclusion of otorrhea is on two grounds: firstly, many recover as a result of surgical or other treatment, noticeably so if the antrum is drained by a postaural operation not too long delayed, so they cannot be classed among the deaf; and, secondly, in the presence of otorrhea or of the initial stage of nonsuppurative catarrh where a partial vacuum fills the tympanum with a serous transudate, functional tests are valueless. A cough, or a change of position of the head, or any factor which changes the position or quantity of the discharge within the tympanum may at once increase or decrease the residual hearing capacity. Within the past year I have seen a patient whose hearing was comparatively good when he stood up, but he complained of being stone deaf in bed. In the erect position the windows of the labyrinth are above the level of the fluid, but when he lies down they are under water.

It is made plain by the above cases that the operation of adenotonsillectomy, as generally practiced, does not always cure or prevent deafness in children.

Some degree of tubotympanic catarrh so invariably accompanies adenoids that it is possible to diagnose the condition by otoscopic examination alone. In a recent publication I made the statement that I had never found a normal drumhead in a child with adenoids. That is an overstatement, for I have since found, on referring to the records of 127 cases examined at the School Aural Clinic, in 1927, and carefully charted, that one child, from whom an enlarged pharyngeal tonsil was subsequently removed, is recorded as having a normal otoscopic image at the time of the preliminary examination. The fact remains that many hundreds of children may be examined in series and a normal drumhead never be observed. I define a normal image as follows:

(1) The membrane has a pearl gray luster with a surface appearance of polish, free from roughness, scars, depressions, perforations or patches of atrophy or calcareous degeneration. (2) The light reflex is at an angle of more than 90 degrees with the long axis of the handle of the hammer bone. The light reflex is unbroken except at the sulcus tympanicus. In 400 children with normal ears, between the ages of 5 and 14 years, examined by the writer, the light reflex did not reach the periphery in 368, or 92 per cent. About a millimeter from the sulcus tympanicus there is in these cases a break in the cone of light due to a change in the curvature of the membrane at the annulus. A continuation of the light reflex beyond the break, when such continuation occurs, is the "sulcus light" of German writers, and is normal. A peripheral discontinuity of the reflex cone of light is therefore not an indication of tympanic disease.

(3) There are no supernumerary light spots.

(4) The short process of the malleus is distinctly visible as an ivory white knob. Extending from this process to the periphery of the drumhead are the anterior and posterior folds which separate Shrapnell's membrane from the pars tensa. The anterior ligament is straight, and while in the adult it is very short (1.5 mm.) and sometimes unrecognizable, it is always prominent in the normal ear of the child. The posterior fold is thrice the length of the anterior (3.5 to 4.0 mm.). It is not straight. It curves upwards, backwards and finally downwards,

to be lost in the annulus fibrosus at the point where this latter is attached to the spina tympanica posterior. These folds in the drumhead are due to the underlying anterior and posterior ligaments which attach the malleus to the tympanic ring and are the axis upon which it moves. The normal curvature of the posterior fold must not be confused with the so-called posterior fold which results from retraction of the drumhead. This retraction fold, which also originates at the short process of the malleus, is not lost in the annulus tympanicus at the posterior tympanic spine, but curves downwards and backwards, concentrically with the annulus, and often reaches the inferior posterior quadrant of the drumhead. Its presence is an indication of disease.

(5) No dilated blood vessels are visible in any part of the normal image.

(6) The handle of the hammer bone is neither foreshortened nor abnormally broad, nor is its line of attachment to the drumhead, the stria malleolaris, unduly prominent. When tested with the pneumatic speculum it moves in an equal degree with the drumhead; but no more and no less. While the normal drumhead moves as a whole together with the malleus, the excursions of its posterior superior quadrant are visibly greater than those of the rest of the membrane, but it does not flap on movement. It is nominally more transparent than the other quadrants, so that the long process of the incus, and even the incudostapedial articulation and the posterior crus of the stapes are frequently visible through it.

(7) The one area of the otoscopic image which is normally opaque in the child is Shrapnel's membrane. The cause of this opacity is a strip of skin which extends downwards on to the posterior section or Shrapnel's membrane from the roof of the external meatus, carrying with it the principal vessels and the nerve of the tympanic membrane. It is the *bande cutanee* of Tillaux, and the *cutis-stranger* of German writers.

These remarks apply only to children of school age. The otology of infancy is a problem apart.

I have elsewhere published the observation that the earliest pathologic changes in the tympanum are reflected in the otoscopic image. Children who are in no appreciable degree deaf, who have never suffered from otalgia, otitis, otorrhea, who

have, in fact, never had a symptom referable to the ear, and whose tubes are quite patent, yet invariably show an otoscopic image which is in some degree abnormal. This in its earliest manifestations is not well understood, for the technical difficulties of obtaining any quantitative estimate of the various factors have until recently proved insurmountable. It is hoped that the advent of the *Ohr-Mikroskop* of Luscher (Berne), which permits of the examination of the living *membrana tympani* under high magnification, and the accurate micrometer measurement of it, will result in important discoveries. This work is now being undertaken at my Aural Clinic, but it will be premature to publish results for some years to come.

From the clinical point of view, which is the one at present of importance, it is not difficult to indicate the otoscopic appearances of early tubotympanic disease. In 477 adenoid children there was one with normal tympanic membranes. In view of the fact that a number of these children in later years drift into chronic progressive deafness of a hopeless type, I consider the above observation to be of some importance. It gives point to my belief that nothing can be of more importance than a study of the adenoid child, in whom we find, and not alone from the otologic viewpoint, many early, but as yet largely unappreciated indications of future disaster.

ADENOID DEAFNESS.

Adenoid deafness as distinguished from the residual deafness of suppurative otitis media, is purely mechanical in origin. Suppurative otitis media, which is either cured or becomes chronic, does often lead to deafness but never to chronic progressive deafness, for when the process of scar tissue formation is at an end the resulting functional deficiency becomes a fixed quantity, and the deafness is not only nonprogressive but is even in some degree amenable to treatment and may be relieved and sometimes cured.

Excluding otosclerosis, whose etiology is unknown, and the various obscure conditions erroneously classed together as nerve deafness because they are found to have a diminished bone condition, adenoid deafness is not only the most frequent variety of chronic progressive deafness met with in practice,

but also the only one preventable in the light of present knowledge and will be alone discussed.

It develops in three stages:

Stage 1. The stage of occlusion.

Stage 2. The stage of transudation.

Stage 3. The stage of cicatrization.

The description of each stage will include remarks upon the resulting otoscopic image.

Stage 1. Occlusion.—The eustachian tube becomes occluded by the pressure of the pharyngeal hypertrophy and the resultant swelling of its mucous lining. This is at first an edema of mechanical origin, but it is soon replaced by a leucocytic infiltration of the mucous and submucous layers, which produces such a degree of obstruction that the tympanum becomes a closed cavity. The oxygen of the imprisoned air is absorbed by the blood vessels of the mucous membrane and a partial vacuum results. The external atmospheric pressure drives in the drumhead, fixes the ossicular chain and produces a middle ear deafness. This stage in children is always accompanied by some degree of earache.

Examination reveals the characteristic otoscopic image of early tubotympanic catarrh: (1) The handle of the hammer is foreshortened and retracted, and the short process is seen in exaggerated relief. (2) The reflex cone of light, normally at an angle of more than 90 degrees with the stria malleolaris, assumes a more horizontal position, and may even be at an acute angle. It is usually broken, sometimes altogether absent, particularly in young children, and if the degree of retraction is great there are supernumerary light spots in other parts of the field, usually above or on either side of the short process. (3) The posterior fold is exaggerated. (4) The annulus fibrosus stands out as a white ring, in marked contrast with the faint peripheral flush due to congestion. (5) The blood vessels which run down the handle of the hammer, posterior to the stria malleolaris, may be markedly dilated, but this is not a constant feature in the young.

The image differs from that seen in the early stage of acute suppurative otitis media. In the latter the landmarks are usually obscured, the drumhead is of a uniform angry red, and the superior posterior quadrant either bulges or is obvi-

ously the seat of an acute inflammatory process. In no stage of adenoid deafness is there bulging of the tympanic membrane. Retraction is characteristic and pathognomonic; bulging invariably means that active infection has changed the condition into suppurative otitis media.

In the stage of occlusion, inflation of the middle ear results in a temporary alleviation of the deafness and discomfort, which, however, invariably return within a few hours or a few days. It is at this stage, and this stage only, that we can with any reasonable certainty, hope for a permanent cure by removal of the pharyngeal hypertrophy.

Stage 2. Transudation.—Within a period which in children is so variable that it may be hours or weeks, the tympanic mucous membrane becomes edematous from the negative pressure and a serous transudate from the lymph spaces and blood vessels partly fills the tympanic cavity. It never entirely fills it, because its quantity, being dependent upon the condition of partial vacuum, its formation ceases when the negative pressure is equalized.

Intratympanic fluid, which is the product of infection, continues, upon the other hand, to be exuded so long as the infective process is at work, with the result that the drumhead bulges and if not drained by surgical intervention usually ulcerates and ruptures.

With the flooding of the middle ear and the resultant fall in pressure, the drumhead reapproaches, but never quite attains, the normal position. It has been shown by Schrebe (1892), Brieger (1896), Launois (1896) and Kümmel (1908) that the transudate is sterile (Phillips). Not only is the transudate sterile, it is bactericidal also, any organisms found in it being in process of disintegration (Alexander). According to Launois, it becomes rapidly sterile in cases where culture during the first few days gives a positive result. This is in exact accord with clinical experience, and accounts for the rarity with which this form of tympanic catarrh is followed by supuration.

With the fall in pressure the capillary engorgement disappears. At this stage it is often possible, particularly with a magnifying otoscope, to observe the fluid through the tym-

panic membrane. It may be recognized by contained air bubbles if the eustachian obstruction is not complete, or as a fine hairline meniscus at either side of the handle of the malleus. That the appearance is due to fluid is shown by the horizontal position which the hairline assumes, whatever be the position of the patient's head. Adult patients are often aware of the presence of fluid in the middle ear, and feel its movements, but children are particularly tolerant of any aural discomfort which does not amount to actual suffering, and rarely complain.

This transudation stage is dangerous, for two reasons. Infection may convert it into a suppurative otitis media, with the possibility of mastoiditis and intracranial spread, and the certainty at least of a troublesome and disabling disease whose prospect of cure is as problematic as its potentiality for harm is unlimited.

In the absence of gross infection leading to suppuration there is still the possibility of the commencement in spite of whatever treatment is undertaken, of the slow process of meso-tympanic cicatrix formation, which is the third stage of adenoid deafness and is to be regarded, for all practical purposes, as incurable.

The treatment of the second differs in no respect from that of the first stage, though it is less likely to be entirely successful. My observation of a number of cases kept under observation for many years leaves no doubt that the commencement of permanent intratympanic changes may be coincident with the appearance of the transudate. When the hyperemia ex vacuo of Stage 1 is followed by the hydrops ex vacuo of Stage 2 the aurist may be tempted to drain the tympanum of its fluid by paracentesis of the drumhead. While this may be theoretically sound, I am convinced that in practice it is a surgical error. It places the sterile tympanic cavity in direct communication with the external auditory meatus, from whence infection is a practical certainty.

Stage 3. Cicatrization.—The organization of the exudate into fibrous threads, the transformation of the infiltrating leucocytes into connective tissue cells, and the hypertrophy of the mucous membrane, which characterizes the cicatrization stage,

may be due to a bacterial infection of great chronicity and low virulence, or may, upon the other hand, be comparable to the formation of aseptic scar tissue elsewhere, a process which we may justly deem to be physiologic. Upon these points I am not competent to judge, and having no evidence to produce, content myself with noting that certain continental authorities, Lermoyez in particular, seem to think that the stimulus behind such processes may be a biochemical toxin due to errors of metabolism. The latter author rather vaguely particularizes the localization of constitutional states upon the mucosa of the upper respiratory tract as an efficient cause, "*en particulier le catarrhe suintant des enfants blonds lymphatiques et le catarrhe hyperemique des adolescents arthritiques a vaso-motricite dereglee.*"

Whatever the pathologic explanation may be, the result is severe and incurable deafness, primarily due to fixation of the ossicular chain by a network of newly formed bands of connective tissue, and the partial obliteration of the tympanic cavity and attic by hypertrophy of the mucous lining and its many folds and extensions. The area of the stapes are bound by fibrous adhesions to the walls of the pelvis, the incudo-malleolar articulation is immobilized, with a consequent disuse atrophy of the stapedius and the tensor tympani muscles, and the now useless drumhead either atrophies and falls in upon the underlying ossicles and promontory, to which it adheres, or becomes the seat of calcareous degeneration. I have seen an almost complete ring of such degeneration, a tympanic arcus senilis, in a deaf child, ten years of age. If in addition to fibrous ankylosis of the ossicular chain the round window of the labyrinth is closed by a shutter of fibrous tissue, deafness for the spoken voice becomes complete.

If the intratympanic processes stop at cicatrization and hypertrophy, the degree of deafness becomes in time a fixed quantity and is no longer progressive. A percentage of these cases, which on account of the lack of adequate following up facilities I am not in a position to state with accuracy, but estimate provisionally between 30 and 40 per cent suffer a further process of degeneration in the form of a secondary atrophy of the hypertrophied tympanic mucoperiosteum with consequent nutritional changes in the underlying bone.

The end result of this is a condition distinguishable from otosclerosis only by the appearance of the drumhead and the previous history of the case.

In true otosclerosis (primary idiopathic degenerative deafness) the pathologic process starts in the bony capsule of the labyrinth, there is usually a complete absence of any history of previous ear, nose or throat trouble, and the otoscopic image is normal, however advanced the disease. In the condition under consideration there is upon the contrary unmistakable evidence of former disease, but the functional disability differs in no way from that of otosclerosis, not even in its incurability. The deafness is usually paracutic and severe, and may be of the tympanic, labyrinthine or mixed variety, while the accompanying tinnitus is frequently intolerable. Every clinical finding indicates a process which is now, notwithstanding the antecedent history, that of otosclerosis.

Sections prepared by Nager of Zurich, and exhibited by him at a recent meeting of the Otological Section of the Royal Society of Medicine, showed true bony ankylosis of the stapedo-vestibular articulation secondary to cicatricial processes within the tympanus, thus confirming the conclusion which I have drawn from the clinical observation of the adenoid child.

FIXATION OF THE STAPES.

Fixation of the stapes is frequent in adenoid deafness. It may be transitory, as when it is due to spasm of the tympanic muscles, or to negative pressure within the tympanum as a result of acute closure of the tube; or it may be permanent as when fibrous ankylosis of the stapedo-vestibular articulation follows an exhausted suppuration, or when true bony ankylosis results from otosclerosis or from atrophy of the mucoperiosteum as in adenoid deafness.

The detection of permanent fixation is of the very first importance to the patient. In its absence there is hope that treatment may at least relieve; if it is present, the prognosis is not only hopeless as regards hearing, but local treatment is likely to make the condition worse, and in true bony ankylosis is contraindicated.

Gellé's test for fixation of the stapes is classical, but it has been found to be of doubtful utility in practice. Results are

not always obtained, and when obtained are not always reliable. I have been interested in this problem for a long time, and as long ago as 1912 published a new method of detecting fixation of the stapes, which has since been republished by Prof. Escat of Toulouse, with modifications which rob it of whatever utility it possessed.

In the same year (1912) I showed that paracusis, which can be artificially produced in every normal ear, is not pathological but is a physiologic phenomena, of which the ability of a certain type of deaf patient to hear better in a noise is but an exaggeration.

My experiments were repeated in a certain number of cases by Dr. (now Sir James) Dundas Grant, who confirmed my results.

The artificial production of paracusis (paracutic reflex) has been made the basis of a useful clinical test. Dr. Charles Heath, late surgeon to the Throat Hospital, Golden Square, and now consulting aural surgeon to the Metropolitan Asylums Board, the inventor of the Heath conservative mastoid operation, and of international reputation, tells me in a private communication that he uses my paracutic reflex as the one reliable prognostic sign in deafness. If the reflex is present he considers the prognosis hopeful, at least as regards the possibility of affording relief; if it is absent he refuses to treat the case.

In children of school age it is very difficult to carry out any tests at all, and yet it is here that the early detection of fixation of the stapes is most important. Gellé's test is not reliable in any case, and it is particularly unreliable in children, whose attention is distracted by the technic to the extent of rendering their responses valueless. The test devised by me in 1912 involves catheterization of the tube as a preliminary, an impossible procedure in a child, and in any case I have abandoned it for a better and simpler one, which will now be described.

It is possible to make an exact diagnosis of fixation of the stapes with no other apparatus than the four tuning forks which I use in the routine examination of every patient, child or adult. These forks are C 34, C 64, C 128 and C 512. The method has proved reliable in a long experience of it. It has been tested in many hundreds of cases, a number of whom have been kept under observation from childhood to adult life.

In no single instance where ankylosis of the stapes has been diagnosed by this method is there any record of any subsequent improvement in the hearing.

The test depends upon two well established otologic principles, viz.: (1) If the labyrinth windows are occluded the lower tone limit is raised in a degree proportionate to the loss of mobility in the windows. That is to say, when the footplate of the stapes begins to move with less than normal freedom the patient cannot hear the vibrations of the C 34 fork by air conduction, but can still hear C 64. When the footplate of the stapes, at a later stage of the disease, has become ankylosed in the oval window, he is deaf to C 64 also.

(2) The second principle upon which this test is based is the fact the amount of loss of mobility in the ossicular chain is indicated by the time relation of air conduction to bone conduction in the perception of tones of varying pitch. A negative Rinne low in the scale (C 64 or C 128) accompanies a mild degree of disturbance in the mobility of the sound conducting apparatus. A negative Rinne high in the scale (C 512) indicates serious loss of mobility in the chain of ossicles. In ankylosis of the stapes Rinne is negative for all forks used in the test.

The practical outcome of this is that if a patient shows a negative Rinne—that is, if he hears for a longer period by bone conduction than by air conduction, when tested with fork C 128, there is some loss of mobility in the ossicular chain. If on testing with a fork an octave higher, C 512, the result is a positive Rinne, that is, if he hears with the higher fork longer by air conduction than by bone conduction, the loss of mobility does not amount to immobility. But if Rinne is negative with both forks, then the loss of mobility in the chain of ossicles is complete and amounts to immobility.

It has been recently stated by Richard Lake that this double negative Rinne indicates fixation of the stapes. But the fact that many patients, particularly children, who show this negative Rinne for both forks, often lose it after operation or other treatment proves that it cannot indicate fixation of the stapes, if by fixation is meant either fibrous or bony ankylosis. This test has been applied by me to every patient before, during and after treatment for many years.

In certain cases where treatment relieves but does not cure the deafness, and these are in a majority where the patient is an adult, the Rinne becomes positive for the higher fork, indicating some return of mobility to the stapes, but remains negative for the lower fork.

In cases of progressive deafness, where treatment has given no relief, it will be found that in addition to the negative Rinne for both C 128 and C 512, there is absolute loss of hearing by air conduction for C 64.

This tuning fork triad of negative Rinne for C 128, negative Rinne for C 512, and absolute loss of hearing by air conduction for C 64 is pathognomonic of ankylosis of the stapes in the vestibular window.

XLVI.

THE PARANASAL SINUSES AS SOURCES OF
INFECTION.*

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There seems to be a constantly increasing tendency on the part of the medical profession to locate the primary foci of many heretofore unexplained focal infection syndromes within the paranasal sinuses. There is voluminous literature today supporting this hypothesis. The writer has been unable to agree with this trend. For several years a conviction has been growing in his mind that this explanation of many focal infection syndromes has been insufficiently substantiated. While admitting that nasal sinus infections are more or less frequently primary foci in focal infections, the exact mechanism of action has been neither clearly explained nor sufficiently stressed. He also believes that the frequency of such occurrences has been overestimated.

It may be pertinent to ask the question, "What is the popular conception of focal infection?" or "What should be the accurate interpretation of the term?" The proper conception of focal infection among even the intelligentsia seems to vary widely. Some appear to regard a focal infection syndrome as a conglomeration of far removed symptoms or disorders produced by a single local infection, without any distinction being made as to whether the secondary conditions are manifestations of anaphylaxis, allergy, toxemia or real secondary infection. Others seem to have a more constricted idea that focal infection is the production of real secondary infection by a primary focus of infection. The writer inclines to the latter interpretation. Hereafter in this paper focal infection may be interpreted as defined in Dorland's Medical Dictionary, as follows: "Focal infection is infection in which bacteria exist in circum-

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scribed confined colonies in certain tissues and from there are sent out into the blood stream."

It seems evident that a single infection may produce far removed lesions and manifestations of disease, which may be either real secondary infection, toxemia, allergy or combinations of all three. For instance, bacteria from a primary focus may be transmitted through the blood stream and produce a secondary abscess as in pyemia. It is possible for absorption of toxins from a nasal sinusitis to produce a toxic neuritis without actual secondary infection of the nerve tissue. Such a process is clearly demonstrated in tetanus and diphtheria. Does it always follow that every local lesion in the body which may be considered focal must have associated infection? May a cancer be decomposed by enzyme action (prior to any real infection by bacteria) and still produce far removed toxic symptoms? It is possible, theoretically, for an antrum containing excessive secretion, produced by noninfected nasal sensitization, to produce a kindred but far removed lesion, such as an allergic urticaria of the skin or asthma. Such terms as "focal toxemias," or "focal allergies," may be coined to represent toxic and allergic conditions which may be focal but not necessarily accompanied by infection.

The entire profession may be in doubt as to the classification of some lesions, as their etiology and many points in their pathology are not definitely known. The popularity of the term "focal infection" can probably be attributed in part to its convenience.

Pathologic conditions of the nasal sinuses will be discussed in regard to the part they play in:

1. The production of secondary infection.
2. The production of toxemia.
3. The production of allergy.
4. The production of constitutional symptoms, such as chills, elevation of temperature, leucocytosis, adenitis, etc.

As suggested by the title, the chief subject to be discussed is the part played by nasal sinusitis in the production of real secondary infection; however, some remarks will be made as to other effects.

If the nasal sinuses produce secondary foci of infection, is it by direct absorption of infective bacteria into the blood

stream (through proximal lymphatics and blood vessels) or does it occur after the pus has left the sinuses and has descended by gravity or by aspiration downward over other structures of the respiratory or gastrointestinal tract, secondarily infecting these parts with which it comes in contact, thence being fed by them into the circulation?

The writer believes that the production of secondary infection by direct absorption of bacteria into the circulation from the nasal sinuses is one of the rarest occurrences in medicine.

The writer also believes that when nasal sinusitis is responsible for secondary infection in distant sites of the body, and dissemination occurs via the circulating fluids, the mechanism of action is usually as follows: The pus leaves the sinus through its natural orifice, it then infects some other more highly absorptive tissue or mucous membrane and is finally fed into the circulation by this second focus. The most common secondary route is via pharyngeal lymphoid tissue, either tonsillar or extratonsillar in distribution. The action of discharges from the sinuses in secondarily infecting adenoids, tonsils and hypertrophied extratonsillar lymphoid tissue of the pharyngeal walls seems unquestionable. The frequency with which secondary infection of the auditory tube, tympanum, larynx, trachea and bronchi occurs from nasal sinusitis is also not questioned. Even the occasional production of pneumonia by aspiration or gravity descent of sinus discharge may occur. He does not believe, however, that many lung infections from the nasal sinuses are hematogenous or that they occur very frequently through absorption by the circulating fluids directly from the intrasinus mucosa.

The production of severe constitutional symptoms, high temperature, marked leucocytosis and severe adenitis by nasal sinusitis is a rare event unless there is also present an accompanying condition, such as a pharyngitis which was produced by secondary infection from the discharge after it has left the sinus through its natural orifice.

Absorption of toxins directly from the nasal sinuses into the circulation in large enough dosage to produce secondary symptoms is much less rare.

The production of allergic products and sensitizing proteins in the nasal sinuses and their subsequent absorption in considerable dosage by the circulation is not infrequent.

These facts are more referable to adults than to children. It seems that many of the explanations offered in this paper for such conclusions may not hold true in infants, and probably the problems involved are entirely different in the young. However, the writer suspects that even in the young the mechanism of action in some cases may be worked out eventually as secondary to lymphoid tissue because of the known predominance of such tissue in infants.

These conclusions are based chiefly upon clinical and anatomic observations.

The clinical observations responsible for the opinions expressed may be briefly summarized as follows:

1. Accurate and careful study of a great number of consecutive cases of uncomplicated acute nasal sinusitis in the adult has revealed the following predominant characteristics:

(a) Remarkable rarity of severe constitutional symptoms and general prostrations except those characteristic of some preceding or accompanying condition, such as influenza, tonsillitis, otitis media, etc. This surprising mildness of general symptoms is very constantly present, even when considerable drainage block with subsequent headache and local pain and tenderness is evident.

(b) No rigors (although slight chilly sensations occasionally occur).

(c) Low temperatures.

(d) Low leucocyte count.

(e) Very slight increase of polymorphonuclears in the differential.

(f) Absence of prominent cervical adenitis.

(g) Secondary infection has rarely occurred with the nasal sinusitis or following it closely enough to establish any connection between the two conditions unless some other more probable focus of infection was also present.

(h) Vertigo is a frequent symptom, especially of acute frontal sinusitis.

(i) Prostration, pallor and other symptoms probably due to toxemia are seen.

(j) Local and reflex pain, paresthesia, hyperesthesias, headache, photophobia, neuralgia and reflex tics are common.

2. Study of cases of uncomplicated chronic nasal sinusitis has likewise shown a surprising absence of severe constitutional symptoms or the development of real secondary foci of infection or manifestations of systemic disease.

Leucocytosis is not mentioned in connection with chronic nasal sinusitis, because many others have noticed, what is so concisely stated by Dr. Judson Deland, that "Systemic disease secondary to chronic infection of the tonsils or sinuses is associated with leucopenia, lymphocytosis and diminished polymorphonuclear cells in about 40 per cent of the cases."

3. Careful observation of cases of chronic nasal sinusitis complicated by hyperplasia, edema and polypoid degeneration of internasal or intrasinus mucous membranes and foul odor of the discharge due to bone involvement has revealed a surprising absence of accompanying secondary infection or infectious systemic lesions. Such constitutional symptoms as bad color, sallow skin, anemia, weakness, poor appetite and below par efficiency are not infrequent.

4. The rarity of severe complications accompanying intranasal surgery in an infected field impossible to adequately sterilize is certainly striking.

5. Acute exacerbations of chronic nasal sinusitis are much less dangerous and less frequently accompanied by severe symptoms than one would suppose.

6. When severe constitutional symptoms, systemic disorders or secondary infections have occurred simultaneously with or closely following an acute, subacute or chronic nasal sinusitis for a connection to be suspected, almost invariably some accompanying condition, such as tonsillitis, inflammation of extratonsillar pharyngeal lymphoid tissue, otitis media or tracheitis, has been observed to either precede or parallel in severity the production of the secondary condition.

Such observations have led the writer to believe that when secondary infection in a distant part of the body is derived from the nasal sinuses it is usually produced by the pus leaving the sinus, secondly infecting other parts of the respiratory tract and then finding its way into the circulation from the second

focus. Time after time a patient has been seen with a persistent nasal sinusitis, possibly a postethmoid or sphenoid condition, and pus streaming down the nasopharynx, and still the individual was robust, healthy, red lipped, noncomplaining and symptomless. Perhaps the tonsils have been removed; suddenly the patient returns feeling achy, running a temperature and feeling badly all over; examination shows a nasopharynx increasingly red, with tender cervical lymphatics and lymph nodes. With a subsidence of the lymphoid tissue inflammation, the patient returns at once to a comfortable condition, in spite of the fact that the nasal sinusitis goes steadily on with no noticeable improvement.

There is not much anatomic evidence of very profuse blood supply (per square millimeter of surface) to the mucosa or bony wall of the nasal sinuses. The wall of a sinus in a living subject, when opened to surgical inspection, nearly always has the appearance of a cold, inert, pale, white affair. The intrasinus mucosa is seldom of a deeper color than a slight pink. This frequently holds true, even when considerable infection apparently exists or just after an acute infection. This is a very constant observation, except in those chronic cases described by Hirsch and others as the catarrhal variety, in which secondary complications, such as hyperplasia, edema and polypoid degeneration of the mucous membrane have occurred or when the process has extended further and involved the bone as an osteomyelitic process. The mucosa of the lower turbinates, septum, etc., is much more vascular, but the inner aspect of nasal sinuses, even the ethmoid labyrinth, has a comparatively gravelike inertness of appearance. In contrast, the faucial tonsil has a profuse arterial supply and an intricate anastomosis of vessels ramifying about in its structure and surrounding the crypts. The tympanum has a profuse blood supply with many anastomoses. The tympanic membrane is supplied by a cartwheel of vessels, and we are told by anatomists that the arterioles fairly jump into the venules without the usual intricate interlacing of capillaries which would be more sievelike, or, one might say, more of a "filter" of infection where more leucocytes could attack the bacteria as they file through the smaller channels in smaller numbers. Does not this anatomic point perhaps

have a bearing upon the regulation of dosage of bacteria absorbed into the general blood stream from the nasal sinuses?

When a secondary focus of infection occurs in a site far removed from the primary focus, it means that dissemination of the infection has occurred through the circulating fluids of the body, i. e., the lymph and blood streams (excepting of course, peritonitis, skin infections and such conditions in which the infection can migrate by reason of the discharge flowing over some surface of being spread by cilia). Also it occurs where the secondary focus is proximal to the primary one so that extension of infection may occur by contiguity of tissue (direct extension). When the secondary infection appears in a distant part and dissemination obviously has occurred via the circulation, it must mean that a bacteremia has been present. It is not necessary to assume that a clinically recognizable septicemia has preceded the onset of the secondary condition. Too many focal infection syndromes occur with no history of a stormy preceding illness. If there was a history of such prodromes as chills and high fever (similar in character to a septicemia) then it might be difficult to eliminate such a possibility. In any case, it does mean necessarily that a bacteremia must have been present preceding the onset of this secondary infection, and also, that bacteria must have been present in the blood stream in fairly large numbers or in very strong virulent form to have survived and fought off the protective body forces along their route and then remained vigorous enough in the end to attack in their new home. That should mean a very profuse absorption and subsequent dissemination from the primary focus.

A toxemia does not necessarily mean an enormous dosage of the absorbed injurious substance. A toxin may be similar to a vegetable alkaloid. One four hundredth of a grain of aconitine is a dose, when much less than that amount of some virulent toxin may constitute an overdose or even a lethal dose. Therefore, it is consistent to say in one breath that secondary infection is rare, following nasal sinusitis, because there is very little anatomical reason to suppose that much absorption occurs directly into the blood stream from the nasal sinuses, and the next instant to state that severe toxemias can occur from absorption of toxins from the sinuses. The difference

between the total area of surface of the nasal sinus mucosa containing possible infection and the middle ears, even with mastoid involvement, cannot be questioned or doubted. Even admitting that there is not the vascular mucosa in a nasal sinus found in the tympanum, it can easily be seen that some toxins may find their way into the circulation from this enormous surface, though it is but sluggishly active; small doses if virulent enough may produce dire results.

Mullin and his associates have demonstrated by animal experiments the possibility of infection reaching the lung through the lymphatics and circulation. They have traced the lymphatic drainage of the maxillary and frontal sinuses by injecting suspensions of carbon and other colored substances, tubercle bacilli, etc., into the sinuses and nose and believe they have demonstrated the lymph drainage occurs via the submaxillary and deep cervical nodes, cervical lymph ducts to the great veins, right side of the heart and pulmonary artery of the lungs. Here we wish to call attention to the fact that Mullin and his associates were unable to get any demonstrable absorption from the nasal sinuses into the circulating fluids of the body (either lymph or blood) until they had traumatized the sinus mucous membrane. This observation goes a long way toward substantiating our contention that absorption from the nasal sinuses is a relatively sluggish affair.

It is not denied that severe systemic symptoms are not seen accompanying nasal sinus infections, but usually these are due to slow and slight absorption of very virulent toxins contained or produced in the sinuses. Mosher believes that the accessory nasal sinuses are splendid locations for the elaboration of toxins. He believes that toxins may be produced without demonstrable presence of pus formation. He has long been suspicious of the white sterile masses of mucous which are washed from antra now and then. He speaks of the kind so often seen in vasomotor rhinitis cases and of his patients suffering from trigeminal neuralgia. He washed about a dram of mucous (white sterile mucous) from the left antrum of a patient, injected it into the peritoneal cavity of a guinea pig and the animal died in twenty-four hours. The autopsy on the pig showed the abdomen and the rest of the body negative except for a slight congestion of the adrenals. The pig was over-

whelmed by a mortal dose of some toxin contained in the mucous. No bacteria were demonstrated in the mucous injected or in the viscera of the pig which could be shown to be introduced by the injection. He asks if this is anaphylaxis or toxemia. The writer has a profound respect for Mosher and for the truth, veracity and scientific accuracy of any idea he may exploit. It is possible that many so-called focal infection syndromes attributed to nasal sinusitis may be proven eventually by further investigation to be manifestations of toxemia or hypersensitiveness instead of real secondary infection.

Some may think that it is all "much ado about nothing" to make distinctions between secondary infection, toxemia and allergy. One might say that focal infection is broad enough to cover them all and why be more specific? As previously mentioned, is it not true that a great weakness of focal infection to date has been this very tendency to make it explain too many idiopathic things. Accurate interpretation of the pathology of a given lesion should make a marked difference in the prognosis, if nothing else. In the course of one of his probably tiresome inquiries into the specific etiology and pathology of a certain eye condition, the writer was very kindly but firmly accused by an ophthalmologist friend of making a distinction without a difference. The case in point was one of detached retina and was referred by the ophthalmologist to eliminate a focus of infection in the ear, nose and throat field. There was no sign of pus in the nose, no evidence of infection in the teeth or tonsils. However, the patient gave a history of hay fever for twenty years. A very neat resection of the nasal septum had been done nearly twenty years before, but in the posterior third of the septum near the floor of the nose was found a polyp the size of a cherry. The antral washings returned clear. Upon the theory that a detached retina might result from an allergic edema behind it, some very tiresome questions were probably put to the ophthalmologist over the phone in an attempt to get an intelligent understanding of what was known about just how such a condition was produced. Was it considered purely an infectious thing, could toxins cause it and lastly could it be allergic? Of course, it is often hard to answer a rapidfire line of questions offhand, for frequently the facts are not definitely known. However, the writer felt he should find

out more than he actually knew about such a condition before he could intelligently render an opinion in this given case. There was no sign of the suspected focus of infection that could be made out, but this allergic condition was very evident, and, if it could be a possible cause, it might explain all. This case certainly illustrates the point. A case may be referred with a request that a definite focus of infection be ruled out and still some other pathologic process may be present which will as easily explain the condition if the examiner only knows the etiology and pathology of the lesion in question. In getting this information about another man's specialty, one may, like a growing child, often ask questions which appear to be distinctions without real differences, but they may turn out all right in the end. While on this allergic question it might be mentioned that Dr. Jas. G. Dwyer, at Montreal, in 1926, in reading a paper on focal infection before the Trilogical Society, brought forth and strongly stressed the idea that allergic activity of the basal protein of bacteria is the real cause of their ill effects and selective action. Whether all of Dr. Dwyer's researches are eventually confirmed or not remains to be seen, but his ideas seem good. We feel supremely confident that his work will establish the fact that allergy is one very potent factor in the production of dire results by many infections.

Dr. Dwyer also brought out the point that the opportunity for infection to become confined under considerable pressure (as in a fibrous tonsil) is one of the most important factors in the production of focal infection by a primary focus. The writer does not often see nasal sinuses totally obstructed for long periods of time, especially in treated cases. Permanent block does occur occasionally and many of the spontaneous cases of osteomyelitis of the frontal bone unattended by trauma are probably due to such a condition in the sinuses. If permanent drainage block of the nasal sinus is rare then this is another argument against such conditions being dangerous foci of infection.

It is not always easy to determine whether or not a nasal discharge is pathologic. Many years before this paper was contemplated the writer began to ask the question "what is pus in the nose?" It is startling to see what some rhinologists unhesitatingly diagnose as pus. It seems that many have for-

gotten that the normal intranasal membranes should not be as dry as a city pavement in August. If there is any secretion to be seen intranasally, no matter what its character, it is pus. Now what is pus and what is not? This is not always an easy problem to settle with the naked eye, it must be admitted. However, the microscope helps some and will certainly show far less chronic purulent nasal sinusitis than some may believe.

The theory has been advanced recently that nasal sinusitis frequently exists in virulent form with no objective signs present, other than a very slightly discernible serous content of the sinus. It is claimed that often these cases are potentially more active and toxic than others showing frank pus and many leucocytes. This may in some degree be true, but it offers the opportunity to draw the dividing line so finely that an ultra-scientific diagnosis can be made on suspicion in any given case, whether normal or not.

The above theory may in a degree be true, but how can the average man tell when an apparently normal sinus contains this vicious type of infection? Cultures do not help much. Injection into animals is almost as useless a procedure because a microorganism to which one species is naturally immune may kill another species. Even in different individuals of the same species, one strain of bacteria may grow and thrive as a harmless parasite to one host, but when transmitted to another prove fatal. "Typhoid Mary," played havoc with her human contacts, but was not clinically ill herself. Walking, apparently healthy diphtheria-carriers harbor Klebs-Loeffler bacilli very deadly to others. Therefore, no matter what biologic characteristics the cultures may show, how can one determine when they are virulent for the particular individual if they do not show evidence of local inflammation in the area which they have selected as their abode? If one speculates too freely he may as well decide that all nasal sinuses are deadly menaces and proceed to remove them from everyone. Therein lies the danger of these conclusions. What is needed is real tangible evidence that can be applied as a useful aid in diagnosis. If a considerable quantity of serosanguinous fluid is seen in a sinus which one could definitely say was abnormal, and if the skin about the nasal orifice is excoriated, showing the irritating nature of something in the nasal secretion and still no frank

pus can be found, then one has something definite which can be clinically visualized.

To illustrate the observation that acute nasal sinusitis with considerable drainage block is frequently devoid of severe constitutional manifestations, such as high temperature and marked leucocytosis unless accompanied by other conditions such as tonsillitis and pharyngitis, the two following cases are presented:

Dr. O. R. S., physician, had a purulent discharge from all the sinuses on the right side and X-rays showed this entire area practically blotted out with density. Beneath his right middle turbinate a large bulla ethmoidalis was seen hypertrophied, glazed and showing a tendency to undergo polypoid degeneration. Pus was pouring from all sinus outlets on the right side and from the middle meatus on the left. He had pain and tenderness in the frontal area, pain through the orbit, back to the occiput and down the right cervical region. Right upper teeth ached and were "on edge." He had persistent morning headache, slight backache, but temperature fluctuated daily between ninety-seven and six-tenths and ninety-eight and six-tenths or occasionally ninety-nine and six-tenths, but he had a normal pulse, also normal differential and an average of seventy-five hundred white blood cells. He had no pyelitis in spite of the fact that he had experienced previous attacks not connected with nasal sinusitis. No arthritis and no secondary foci were present. He was hospitalized because of his severe headache. In the writer's experience this is a typical average picture of an uncomplicated very severe nasal sinusitis with partial blocking of drainage from some cells and probably complete blocking of others. Dozens of cases similar to this have been witnessed the past winter (1925-1926) during a rather severe influenza epidemic complicated by secondary infection with an especial affinity for the nasal sinuses, throat, middle ears and mastoid.

Mrs. Lulu D., a nurse, 29 years of age. The writer was called to see this young lady, ill in bed, complaining of a constant unbearable headache, both day and night. Duration forty-eight to seventy-two hours. She was of a very thin wiry type with sharp pinched features, but absolutely nothing could be found except a slight suggestion of swelling and redness of

the nasal mucous membrane. Temperature was subnormal (ninety-seven by mouth and ninety-seven and six-tenths per rectum), pulse in the eighties. A tentative diagnosis of blocked nasal sinusitis was made, more by elimination than anything else. No history of preceding rhinitis could be obtained. Her headache was so severe that she was sent to a hospital, where consulting internists suspected a tuberculous meningitis until a negative spinal puncture was made. White cells were eight thousand. Nothing but nasal treatment was instituted for about forty-eight hours when a profuse purulent post-nasal secretion burst from the posterior group of sinuses on one side at which time the headache was instantly relieved and the patient wished to leave the hospital and return to her work as an office assistant to a physician. She was advised, however, to remain in the hospital for several days. Two days later the hospital resident called and reported that she was again ill, but this time with a temperature of 101, general achiness and leucocytosis of 15,000. Examination showed an acute lacunar tonsillitis, adenoiditis and severe general infection of the extratonsillar lymphoid tissue on the posterior pharyngeal wall. She made an uneventful recovery after her throat infection cleared up.

The striking features of this case were the subnormal temperature and lack of leucocytosis all during the course of a nasal sinusitis which was completely enough blocked for four or five days to produce a headache simulating a meningeal type in severity.

Perusal of case records of acute sinusitis, in the writer's collection and in those of his office associates, have shown case after case of severe acute infection with no cervical adenitis, no, or only slight elevation of temperature, pulse or white blood count and with only slight or no increase of polymorphonuclears in the differential; very little general achiness or other constitutional evidence of great toxic absorption, and without the slightest manifestation of any secondary foci of infection. Of course localized pain, headache and neuralgias are frequent complaints when there is blocking of drainage or severe inflammatory intrasinus reaction accompanying the sinusitis. It has been a very constant observation that rarely do secondary foci of infection occur during or closely enough following nasal

sinusitis to establish any connection between the two. Especially is this true if the nasopharynx and pharynx are free of prominent lymphoid tissue.

Otolaryngologists are often called during influenza epidemics to see cases with extremely high temperatures and asked to explain this fever in the absence of other positive findings by sinus infection. Perhaps a nasal sinusitis can be seen to be present, but on the next visit to another bedside one will probably see just as sick a patient with 105 temperature and no nasal sinusitis. The high temperature in both instances is due to a general constitutional reaction to the influenza infection and the nasal sinusitis when present plays only a minor part in its production.

In the papers of Dean and Byfield, Dean and Armstrong, Jones, Arbuckle, Mullen, White, Marriott and others, when illustrative cases are reported, usually the tonsil and adenoid situation is carefully gone into, but very rarely is any mention made of the condition of the lymphoid tissue on the postpharyngeal wall, lateral pharyngitis, etc.

Below are brief sketches of two typical cases illustrating the role of a posterior pharyngitis with much hypertrophy of the lymphoid elements on the posterior pharyngeal wall exacerbating and producing marked constitutional symptoms, evidently caused primarily by nasal sinusitis.

The record of Miss M. L., aged 12, is submitted to demonstrate the action of pus from an acute exacerbation of a chronic nasal sinusitis secondarily producing an inflammation of extratonsillar lymphoid tissue after tonsillectomy, which in turn produced unilateral cervical adenitis. Her tonsils had been removed December 28, 1925. In the fall of 1926 she had two or three attacks of acute nasal sinusitis, each of which after a few days, persistently localized in her antra. Her post-nasal space constantly showed a yellow nasal discharge coming backward through the choanæ with subsequent redness, enlargement and irritated appearance of the lymphoid tissue on the posterior pharyngeal wall and especially the right lateral pharyngeal band. On January 22, 1927, following her last acute attack, she appeared with a marked enlargement of the right upper cervical glands, chiefly anterior to the sternomastoid muscle, but also some glands affected posterior to this

muscle. A very marked lateral pharyngitis chiefly of the right side was evident. With subsidence of the pharyngitis the adenitis improved accordingly although the postnasal discharge persisted with no change for some weeks. The antra were not permanently drained because the child was not severely ill and the mother did not wish her to miss the time from school.

The record of J. L., a boy, 11 years of age, is cited to illustrate that nasal sinusitis discharges may produce nephritis by secondarily infecting hypertrophied extratonsillar lymphoid tissue which in turn apparently incited the nephritis. He had been subject to nasal infections, sore throats and middle ear infections since birth. His tonsils and adenoids had been removed November 9, 1916, when he was three years of age, but no improvement in the frequency of sore throats, nasal sinusitis or middle ear infections resulted. A marked hypertrophy of extratonsillar lymphoid tissue progressively increased. The lymphoid tissue piled up in such tumor like masses that portions of it were finally snared off and removed. Throughout all his years of trouble high fever, severe constitutional symptoms and prostration in bed always paralleled inflammation of extratonsillar lymphoid tissue of the pharynx regardless of the onset, course or cessation of the nasal sinusitis. Finally, in 1924, when the lad was 11 years of age, he developed an acute hemorrhagic nephritis, following a pharyngitis which in turn followed a severe nasal sinusitis. With treatment the nasal sinusitis improved, but the nephritis gradually abated only after snaring off large masses of the hypertrophied lymphoid tissue which approached tonsillar tissue in volume and mass.

Sometimes this pharyngitis precedes the nasal sinusitis and sometimes a tracheitis, bronchitis, or laryngitis may precede both. A respiratory infection seems to spread in all directions, up or down; however, spreading from the sinuses downward is obviously an easy way because of aspiration and gravity. A nasal sinusitis is usually slow to be entirely erased and will undoubtedly keep the lower fires burning, when if they were not fed by reinfection from above, they would storm a few days and then burn out much earlier.

May 24, 1926, another young man of about 15 years of age, was seen at his internist's request. Internist reported every finding in his field negative for explanation of a rather recur-

rent or septic type of fever of one week's duration. No rigors. Patient was seen at 5 P. M. with temperature of 103 plus. Internist suspected the nasal sinuses. There was an evident oncoming nasal sinusitis, nose perfectly free, but red and juicy, posterior pharyngeal wall only slightly glazed and lymphoid tissue evidently enlarged (tonsils well removed), but the trachea and larynx were red and inflamed, voice husky, cough painful. Although no rales in the chest the site of the infection was evidently the larynx and trachea, probably influenzal in origin. Again the nasal sinuses were not the explanation of the temperature primarily. His headache was in the afternoon, when he had an elevation of temperature, never the morning type. This same patient had been seen many times with a much more severe nasal sinusitis and had come to the office noncomplaining. His reaction to all types of nasal infection in the past was well known. He was a chronic hay fever sufferer as was his brother, sister and mother. A trachea or larynx will react violently, whether preceding or accompanying a nasal sinusitis.

From personal experience the writer has for many years worked comfortably and uncomplainingly through one or more nasal sinus infections without fever or aches. At times he has developed a tracheitis or laryngitis and then has always felt achy with slight fever because he has normally a low fever, but is always toxic in these attacks. Often the tracheitis precedes the nasal sinusitis by from three to five days. Sometimes it is the other way round, but his toxic symptoms always parallel a lower infection of the respiratory tract than the nose.

Great numbers of case records of chronic nasal sinusitis, some of them of years' standing with polypoid degeneration, foul odor and osteomyelitic processes of the bones containing the accessory nasal sinuses, have been carefully searched and studied. It is astounding how many of these individuals were healthy, robust and normal in every way. Real secondary foci were not found in these individuals with the frequency that they theoretically should obtain in view of the purulent character and extent of the pathologic processes. Toxic aches, usually of more or less transient nature, were seen much more frequently, but the production of real secondary foci of infec-

tion was not the rule. The allergic cases complicated by chronic sinus infection showed the most marked emaciation and systemic depression. There was a marked prevalence of anemia and bad color in some of these cases, probably due to the hemolytic nature of some toxin produced in the sinuses.

Mr. C. A. N., male, of 42 years, a large, healthy man of 180 pounds, who said he had scarcely had a complaint in his life except a foul purulent nasal discharge and intermittent obstruction to nasal breathing on the left side, which had persisted since childhood. His nose had been operated upon several times previously.

Left nostril was completely packed with polyp coming chiefly from the middle meatus. Very profuse foul purulent nasal discharge from both middle meati and a few polypi beginning to appear in the right nostril.

Physical examination was otherwise entirely negative. Blood pressure normal, urine clear. Heart and lungs negative. No arthritis and no other focal infection or toxic condition could be found.

Nasal polyp were removed, both antra were operated and the nasal condition was much improved, but his general condition was perfect prior to operation.

Mr. H. W. L., male of 47 years, a large robust man weighing over 200 pounds, with cheeks like a baby, who gave a history of nasal sinusitis for as long a period as he could recall. Both nostrils were practically blocked by polypi coming from the middle meati and also above the middle turbinates. There was a profuse bilateral, very purulent, nasal discharge of a terrible odor. He had recently taken out life insurance and been carefully examined. Blood pressure about 120, heart and lungs negative and urine clear. He has never suffered from arthritis or any other focal infection or toxic complaint. Polypi were removed, both antra operated and the intranasal result was very satisfactory to the patient, but improvement in his general condition prior to operation was impossible.

Dr. L. W., dentist, 27 years of age, had been having repeated reinfections of his nasal sinuses for two or three years following a severe influenzal infection. Had been steadily losing weight, strength and color. Generally below par in efficiency

and appetite. Frequently had morning headache. No arthritis or symptoms of secondary infection.

Examination, September 1, 1924. Slight build, color very bad, anemic and rather septic in appearance. Nasal septum deviated to the right. Pus streaming from right middle meatus. Tenderness over right antrum and frontal. Left middle turbinate large, apparently containing a cell.

In spite of a thin, emaciated appearance, his general physical examination was negative and no contraindication to operation found. Temperature ninety-six, pulse ninety, respirations eighteen.

Operation, September 2, 1924. Submucous resection of nasal septum, removal of left middle turbinate and intranasal drainage of right antrum.

Subsequent course.—Rapidly regained normal health, color and vigor. Gained twenty pounds in two months. He recently wrote that he was too busy to come to St. Louis for observation, but was well and felt that he would need no further intranasal surgery.

Comment.—He had no evidence of actual secondary infection, but his anemia, emaciation, general loss of weight and strength was evidently due to absorption of some toxin from his sinuses.

Mr. A. F., aged 46 years, coal miner and notary public, came to the office complaining of chronic nasal catarrh, obstructed nasal breathing and hay fever every August for many years. His wife complained of the odor from his nasal discharge. He had suffered with morning frontal headaches and occasional dizzy spells. He had been feeling below par physically for a year, had noticed his color getting bad, appetite not good and not so efficient in his work.

Examination.—A fairly muscular individual of medium height and weight, but color much too pale, evidently below par in hemoglobin and red cells. Both nostrils were almost obstructed by a very irregularly thickened and deviated septum and nasal polypi coming from various places. The nasal mucosa was moist, juicy and boggy, pitted momentarily when probed and was of a pale livid color. There was a foul odor and a yellow purulent stream of pus from the right frontal area. The right frontal opening had already been thrown into

one large cavity with the anterior ethmoid cells and a probe entered the sinus easily, passing through a mushy debris of bony spicules. There was almost complete decalcification and softening of bony partitions of the anterior ethmoids and the anterior portion of the middle turbinate. The writer had never before seen such bone destruction in this area except in two cases of lues. The Wassermann was negative. In spite of the negative Wassermann he was put on mixed treatment of mercury and iodides immediately.

The septum was resected, both antra permanently drained intranasally, all anterior groups of ethmoid cells on the right side exenterated and the nasofrontal duct markedly enlarged.

This proved to be one of the very few entirely successful intranasal frontal operations that had fallen to the lot of the writer. Healing was rapid and uneventful, all odor ceased, and there is still a large opening into the right frontal sinus through which the largest probe can be passed. No evidence of polypi dropping down from the sinus. Large intranasal antral openings are still patent, and there has not been enough return of polypi to require further surgery in over three years. His nasal discharge is no longer foul. Nasal breathing is satisfactory and he has missed hay fever for three successive summers. His color and general condition rapidly improved following the operation, and he has remained well and efficient in every way since. Of course, there are other years yet to come, but so far the result has been a very agreeable surprise.

This case is reported chiefly to illustrate that nasal sinusitis in the presence of an allergic intranasal condition frequently produces toxins or some substances probably of hemolytic nature with resulting secondary anemia, emaciation and general inefficiency due to their absorption.

There is an increasing tendency to send obscure cases of infection to the rhinologist for explanation by nasal sinusitis diagnoses. Arthritic cases, high temperature, profound illness (without evident cause), endocarditis and other conditions almost beyond number are being referred for explanation by positive sinus findings. The writer firmly maintains that there is almost invariably some other cause to be found. Especially is this true in either acute or chronic cases if the tonsils have been removed and subsequent compensatory hypertrophy

of the extratonsillar lymphoid tissue has not occurred. The lingual tonsils should be mentioned here also, as a lingual tonsillitis is not so rare as one might think if a careful routine examination is made in all cases of unexplained temperature. However, given a case of perfectly normal nasopharynx, oropharynx, laryngopharynx with removed tonsils and an admitted nasal sinusitis, very little constitutional reaction is ever noted and practically never secondary foci of infection unless otherwise explainable. Usually when extremely high or persistent temperatures are sought for in the nasal sinuses the cause is not there; a central pneumonia, an endocarditis, pyelitis or some other condition will eventually be found to have been present (perhaps only in its incipency) while the nasal sinuses were being searched for the explanation.

It is not our intention to deny absolutely the possibility of secondary infection occasionally occurring by direct absorption into the blood stream from the nasal sinuses without first leaving the sinus to enter elsewhere. Occasionally (usually in children) we so see cases of spreading meningitis in which the infection is apparently catapulted into the intracranial tissues and very probably from the nasal sinuses. Sometimes it occurs within twenty-four or forty-eight hours of the noticeable onset of illness. However, in all such cases careful examination should be made to eliminate abnormal ear drums, or throat and lung complications. It is probable that when great absorption of bacteria from the sinuses by the circulation occurs in adults, it is due to some rare combination of extremely virulent organisms which help each other to extend or in some way to bring about a liberation from localization. We know how deadly some combinations may be when acting together; for instance, the hemolytic streptococcus and pneumococcus combination with an epidemic influenzal infection. Perhaps one organism softens and causes unusual degeneration of the normal sinus mucosa and bony wall with erosion of blood vessels channels, etc., allowing other bacteria of a migrating nature to get out and into the circulation. Even the theory of gas production by bacteria, causing increased intrasinus pressure with consequent bursting forth through weakened sinus walls is tenable. Dehiscences, congenital or otherwise produced, and abnormally thin sinus walls (theoretically, at least) can explain how easy

cellulitis, orbital abscesses, etc., may occur in some cases with consequent difficult localization and easy dissemination. In other words, the true explanation is that the spread almost invariably has occurred from some secondary complication of the sinus condition. If the sinus infection has eroded bone, after passing or destroying the mucous membrane, and osteomyelitis of the surrounding osseous structures has been produced, the secondary infection may occur. However, here the process is different. This is not any longer nasal sinusitis *per se*, but a complication usually diagnosable. It is at that time unlimited in its extent of spread or of body absorption and dissemination of either bacteria or toxins, although such complications are more rare than one would suspect. It is then possible to erode small or large vessels and lymphatics and get into the circulation for a general spread. However, the above condition is a complication which is not easily overlooked and usually admits of no argument. It is too pronounced and too profound a condition to be offered as the explanation of so many silent unsuspected causes of infection which are usually being sought for when puzzling cases are sent to the rhinologist for treatment of nasal sinusitis. Such conditions are not frequent when the total number of infected sinuses are considered and, when seen, are usually found in the comparatively young. Here most of the perforating frontals, ethmoids, orbital abscesses, meningitis, cellulitis secondary to nasal sinusitis occur.

Nasal sinus infections unquestionably do great harm to adjacent structures such as eyes, orbits and brain and also to the lower portion of the respiratory tract. The relation between the nasal sinuses and many eye conditions is purposely left out of detailed discussion because of the chaos and conflict of ideas in the writer's mind at present as to how best to handle the subject. He is uncertain whether the preponderance of evidence is really for or against focal infection, and he is in doubt as to how to explain some of the really remarkable results that he must admit having seen follow nasal treatment and operation in eye conditions. He does not believe that all of these conditions are caused by infection within the sinuses. There seems to be some definite connection between the nasal sinuses and many eye conditions which has not as yet been

clearly explained. However, many of these relations apparently have nothing to do with infection. It is more probable that some of them have to do with abnormal anatomy or growth of the sinuses and are mechanical in nature rather than due to infection. The vacuum theory of Sluder recently elaborated by Dr. Leon White, of Boston, in a very important contribution entitled, "The Influence of Negative Pressure in the Sphenoid on the Optic Nerve," may be tenable.

Two recent cases, one of which was a diplopia due to a sixth nerve paralysis and the other a severe trigeminal neuralgia with signs of third nerve irritation and beginning paralysis on the homolateral side, presented enormous sphenoid and postethmoid cells. These sinuses in both cases were perfectly dry and pale as marble when opened, no secretion, no redness, no swelling of the mucosa, no enlargement of any vessels, could be detected with the naked eye. They were very large and had the appearance of blown-up balloons, as though their bony walls were dilating outward in all directions in their growth, or as if they were being pushed outward by some invisible inward force. Not the slightest sign of any active or passive recent acute or chronic inflammation could be determined with the naked eye at operation. Both cases, however, made remarkable recoveries beginning immediately after operation, though they had been going progressively from bad to worse prior to operation.

A third case was one of beginning optic neuritis getting progressively worse for a week. The patient awakened one morning with blurred vision in one eye, and became practically blind in two or three days. He had a badly infected kidney, since removed. The urologist denied that the kidney was blocked sufficiently to produce the eye symptoms. The case was sent back to the writer. We had just discharged the patient as well of an acute sinusitis about three weeks before. Thinking that in spite of a normal appearing nose there might be more trouble remaining following the recent sinusitis than the evidence indicated, the sphenoids and ethmoids on the side of the lesion were unhesitatingly opened. If no one else would take the responsibility for the condition it seemed unwise to take a chance of allowing the boy to go blind for want of a sphenoid and ethmoid operation—(antra had been explored and found negative). This could be done without great danger

even if no good was accomplished. His sphenoids and ethmoids on the side of the blind eye were operated on. The sinuses were normal in every way. They were a little larger than usual, but not of the extremely large size just described in the other two cases. Within three days the patient began to notice a difference in his vision, although the oculist could not detect improvement. Within another day or two he began to count fingers accurately at ten to twelve feet and within a couple of weeks was reading large type and within a month reading within a line of normal at 20 feet.

Whether the results in such cases are to be explained by counter irritation due to the operation, removal of negative or positive pressure within the sinus, disturbance of lymph flow or some other circulatory phenomena is hard to say. One case might be a coincidence, but nearly every rhinologist can report similar cases.

Evidence of secondary infection of lower portions of the respiratory tract from nasal sinusitis is too absolute to be denied. Such occurrences are too frequent to be overlooked. The incidence of secondary bronchitis, bronchiectasis and even lung abscess complicating chronic nasal sinusitis has been brought out by W. V. Mullen and others in excellent papers.

Rist and Sargent, in 1916, reported observations of supposedly tuberculous soldiers in France and found mistakes in diagnosis in about one-third of the cases regarded as pulmonary tuberculosis and in most of these cases the lung infection was found to be due to paranasal sinus disease.

Zarniko states in his textbook that many times a stubborn putrid bronchitis simulating pulmonary tuberculosis can be attributed to aspiration of sinus pus.

Hajek recommends elimination of sinus conditions in preference to pilgrimages to resorts in a long chain of catarrhal symptoms involving the larynx, trachea, bronchi and lungs.

New has noted metastases of malignant tumors of the antrum to the parotid, submaxillary and cervical lymph nodes. Here, however, is a different problem, as malignancy shows no boundary lines and possibly may invade and extend in various ways from any place. This occurs more slowly in some locations (intralaryngeal cancers) than others.

Dissemination of cancer is not necessarily selective in regard to attacking lymphatics, blood vessels or other tissue although one type of malignancy may spread by lymphatics, another by blood vessels and so on. However, the fact that a cancer eats its way through a sinus mucosa easily does not indicate that an infection can do the same.

The work of Mullin seems to demonstrate the possibility of infections in the nasal sinuses reaching the lung through the lymphatics and blood vessels. However, it is one thing to demonstrate a possibility on lower animals and quite another to verify it as an actual happening pathologically in a human being. It is hard to find in the literature definite evidence of embolic lung infection from nasal sinusitis. The consensus of opinion seems to be that lung infection from nasal sinuses is usually by aspiration. However, when Mullin says he can demonstrate the hematogenous route of lung infection from nasal sinusitis in patients it should certainly be accepted.

There has been much discussion of the role of nasal sinusitis in the production of asthma and hay fever. The connection between asthma and nasal allergic conditions, such as the seasonal hay fever types, rose colds, etc., as well as the perennial types known as vasomotor rhinitis, hyperesthetic rhinitis, etc., has been noted for a long time. In the literature it is being emphasized more and more that both seasonal and perennial nasal allergic patients usually have infected antra. Perhaps other sinuses are also involved, but almost invariably the antra are found infected. Owing to the fact that there is usually a hereditary tendency brought out in the family history of most allergic patients it seems probable that the nasal sinusitis is secondary to the other condition. It is reasonable to suppose that eventually the constant irritation of an allergic condition would lower the resistance of the intranasal tissues. After the sinus infection occurs it becomes in itself a local irritant. Therefore, the hyperesthetic rhinitis becomes more severe and thus a form of vicious circle is established. Operation and drainage of the sinus infection produces a marked improvement in the allergic condition as it is the most vulnerable point of attack in breaking the vicious circle. The writer believes that nasal sinusitis can be either the primary or secondary condition because occasionally a patient appears with an abso-

lutely negative history of previous nose trouble or of any asthma or hay fever in his family: who states that his allergic conditions dates definitely from an attack of "flu" in 1917 or 1918. Such cases almost invariably show infected antra and with permanent drainage of the maxillary sinusitis they improve remarkably. The asthmatics are more intractable than the nasal cases, but occasionally remarkable results are reported following nasal treatment in the lower respiratory tract allergies.

Finally, it may be emphasized that the scarcity of systemic infections following nasal operation is certainly striking. This often holds true even when considerable drainage block should result from tight intranasal packing following operation. It is true that the relative amount of blood supply to the intranasal mucosa does not always explain why nasal sinus operations are not followed by more fatal complications, because more vascular intranasal tissues are usually penetrated in entering the sinus surgically. However, there is usually considerable postoperative rise of temperature after intranasal operations. In the writer's experience this is often in some rough proportion to the amount of vascular tissue denuded or to the amount and tightness of postoperative packing. However, this more vascular tissue is usually left widely open and even with considerable packing there is really little opportunity for tightly confined pocketing of infection except in some area lined with intranasal mucosa or bony wall. The writer wonders how much a local concentration of the protective fighting forces of the body has to do with the high resistance to infection in some tissues where absence of profuse circulation is not an adequate explanation.

SUMMARY AND CONCLUSIONS.

1. Absorption and dissemination of bacteria by the circulation directly from the nasal sinus mucosa in large enough dosage to produce secondary infection is not a frequent occurrence.
2. Absorption and dissemination of toxins directly from the nasal sinuses in large enough dosage to produce symptoms is a much more frequent occurrence.

3. The production of severe constitutional reaction and symptoms by either acute, subacute or chronic uncomplicated nasal sinusitis is rare.

4. Blood borne secondary foci of infection produced by nasal sinusitis, result from the pus leaving the sinuses through their natural orifices; descending by gravity or aspiration over other structures of the respiratory tract, such as tonsils, extratonsillar lymphoid tissue on the pharyngeal walls, trachea or bronchi, and secondarily infecting these structures through contact; thence fed by them into the circulation.

5. It is possible that dissemination of infection through the circulation directly from the paranasal sinuses does occasionally occur, probably due to a rare combination of virulent organisms helping liberate each other from localization for extension and spread or else due to abnormally thin walled cells, dehiscences or traumatic breaks in the normal sinus walls.

6. Focal infection with primary foci in nasal sinuses is more frequent in the young than in the adult and is a different problem altogether in such subjects.

7. The lack of great absorption from the nasal sinuses is probably due to the anatomical fact that the sinus mucosa is not vascular and further to the rarity of complete blocking of sinus drainage with subsequent intrasinus pressure.

8. There is some vague heretofore unexplained connection between certain eye lesions and the nasal sinuses. However, it seems to be equally definitely established that focal infection is not the true etiologic explanation in many of these conditions.

201 HUMBOLDT BLDG.

XLVII.

THE EFFECT OF THE FOOD ELEMENTS UPON THE SPECIAL SENSES.*

BY WILLIAM WESTON, M. D.,

COLUMBIA, S. C.

General medicine covers such a big field that it is beyond human capability for an individual to master it all. Therefore, it has been wisely divided into various subdivisions in order that our tasks may become more comprehensive and enable us to do better work. In order that we may accomplish this object it is necessary that we learn some subjects fairly thoroughly and others that we should completely master. The basic subject creates the cleavage in the medical profession, the common ground upon which we must all stand.

Such subjects as anatomy, physiology, chemistry, histology, bacteriology, pathology and all those branches which have from time immemorial been considered fundamental have from time to time been added to and have become basic. Among the most important of the recent additions have been physiologic chemistry and biologic chemistry. These subjects have in turn become incorporated into the science of nutrition. It is the developments in this science that we must diligently follow, because from the standpoint of human interest the most fundamental of all the sciences. Regardless of what branch of medicine we have cast our lot or where we have directed our interest, there are two conditions—growth and development—that must always remain subjects of our concern. All the conditions and factors that influence these processes are an integral part of the science of nutrition.

Developments in this science have thrown so much new light upon our problems that our views have necessarily undergone many changes. Particularly is this true as to etiology and pathology. Experience has taught us the limitations of bac-

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teriology, that we must look to some other source for a satisfactory explanation of many of the disorders and diseases that we meet with. We have recently learned a great deal about the circumstances under which infection takes place and the factors that determine the kind and nature of infection. In other words, we may so arrange the diet as to determine what kind of an infection a person is subject to. This knowledge has brought us to the parting of the ways, where we must determine for ourselves whether we shall be contented to treat defects and end results by mechanical or unnatural measures, thus making of ourselves more or less skillful artisans, or whether we shall prevent or treat them in accordance with our newer knowledge and make of ourselves scientific practitioners.

Yours, like the other groups composing our profession are, or should be, interested in preventive medicine and should practice it. It is what the public does and has a right to expect of you. The field to which I invite your attention presents opportunities that have thus far not been cultivated except in a few individual and outstanding cases.

If you feel the obligation of making your practice conform to this standard and would allow the science of nutrition to assist, you must diligently study the chemistry and physiology of the various elements that compose the diet. You must understand, for instance, that when it is said that a food contains a definite percentage of protein, little thought is conveyed as to its nutritive value, because the food might be of high protein content and possess little value. The value of proteins depends upon the content and variety of amino acids they yield on digestion. The more nearly these proportions correspond to the content of amino acids in the tissues of the animal, the more effectively can food proteins be transformed into body proteins. There exists great variation in the composition of proteins from different sources. Some contain few amino acids, while others contain many. There also exist in the amino acids marked differences in chemical structure and in nutritive functions. Some of the amino acids can be made in the body, as, for instance, glycine, and it is therefore not necessary that glycine radicals be furnished in the protein. On the other hand, tryptophane, an essential protein of body tissue, is not synthesized in the body and must be furnished in

the food protein. This is also true of lysin, which is necessary to promote growth in the young and provide maintenance.

It is likewise important that you divest your minds of the habit of thinking of the ash constituents of foods as a substance. The ash consists of about fifteen different elements, each with its own function and of definite significance in the process of nutrition.

All the food elements, when present in the diet, work in perfect harmony, but when one of them is absent for a time or is present in insufficient quantity over a more or less definite period the harmony is broken and the human mechanism fails to function in a normal manner. The kind and degree of defect that will become manifest will depend upon the element that is absent or deficient and the length of time of such absence or deficiency.

We must also remember that the glands composing the endocrin system are without exception dependent upon the food for such elements as will enable them to function. Notable but by no means isolated examples of this influence are: the thyroid, which depends upon iodine furnished by the food supply. If supplied in insufficient quantities there occurs an enlargement of the gland, which is known as goiter, and various symptoms, the most important of them referable to the nervous system. The other instance referred to is when an insufficient amount of vitamin B is furnished in the food supply there occurs an increase in the size of the adrenals with a consequent disturbance of the secretion of epinephrin. When the amount of epinephrin falls below normal a condition of hypotension results in consequence of general vascular dilatation. We believe that a deficiency in vitamin C results in a disfunction of these organs also.

We are not aware of any satisfactory explanation why, from a deficiency of vitamin B there should result an enlargement of the adrenals, but a decrease in the size and weight of such organs as the thymus, testes, spleen, ovaries, pancreas, heart, liver, kidneys, stomach, thyroid and brain. How many of us have attributed a disorder of any of these organs to a vitamin B deficiency?

It has been observed that when these dietary factors that influence or control growth and development are interfered

with by a deficiency of one element and excess of another, nature does not adjust the inequality and the normal processes are interrupted. To this rule the special sense organs are not excepted. It is also true that a deficiency of several of the food elements may be responsible for a defect of a special sense, all operative at the same time but in a different manner. For instance, a deficiency or disproportion of calcium and phosphorus and a deficiency of vitamin D may cause a malformation of the malleus, incus and stapes, thus resulting in deafness, or should there be an excess greatly above the normal of vitamin B over vitamin A in the diet there may be a proliferation of lymphoid tissue sufficient to block the air from the eustachian tubes and cause them to collapse. If this tissue is scraped away, hearing will be temporarily restored. If, however, food containing a large amount of vitamin A is not given, the lymphoid tissue will recur and will continue to grow until the drum membrane will finally fail to function.

It seems highly probable that any dietary deficiency, whether protein, vitamin or mineral, which is sufficient to interfere with normal growth and development will react unfavorably upon the organs of special sense.

McCarrison and others report that a deficiency of vitamin A in the dietary results in a weakening of the body and increase susceptibility to many infectious diseases, especially of the eyes and ears. It has been repeatedly observed that rhinitis, conjunctivitis and otitis media are of frequent occurrence in children whose diet is markedly deficient in vitamin A. This vitamin exercises the same controlling relation to xerophthalmia as does vitamin C to scurvy. It must be understood that for the production of xerophthalmia this vitamin must be absent or markedly deficient for a considerable length of time. When deficient for a short length of time following the exhaustion of the storage supply there will occur milder manifestations involving the eyes. In this connection it is interesting to note that in children vitamin A deficiency usually manifests itself by involvement of the eyes and in adults the air passages or lungs.

Dr. Grenfell and others have informed us that the peculiar condition which we know as night blindness, or nyctalopia, is common among the people of Newfoundland. A study of

the diet of the people of that country shows a marked deficiency of vitamin A. In Russia, Italy and other countries where the Lenten fast is strictly observed, corneal ulcers and xerosis of the conjunctiva have been often observed.

Doughty, in his book "Travels in Arabia Deserta," has called attention to the many diseases involving the eyes in those tribes whose food supply is both restricted and deficient. He also observes the rather common occurrence of carcinoma in the Arabians living under the same conditions.

It is of interest to note that in certain districts of India, where nyctalopia is especially prevalent, the popular remedy for the treatment of the condition consists in poulticing the eyes with an exudate of fresh goat's liver and the inclusion of liver in the diet.

When vitamin B is deficient in the diet there occur a loss of appetite, restlessness and failure to grow. If the deficiency continues the nerves become involved and there develop polyneuritis in animals and beri-beri in man. We now recognize beri-beri to be due solely to vitamin B deficiency, and all who have seen this disease will at once recall that the organs of special sense become involved.

Among the important manifestations of regression of the nerves supplying the organs of special sense may be noted hoarseness and aphonia, disturbance of the optic nerves, central scotoma and amblyopia, and paresis of the nervus abducens, which is the third of the eye moving nerves.

Among the other important manifestations of vitamin B deficiency is atrophy of lymphoid tissue. When an excess of lymphoid tissue is found in the nasopharynx and throat, we may be sure that there have existed in the diet a relative deficiency of vitamin A and an excess of vitamin B.

Among the manifestations observed in vitamin C deficiency are minute hemorrhages into the taste buds, producing either an absence or perversion of taste. At times there occur hemorrhages of varying degrees into the ocular conjunctiva, the orbit or the eyelids. When hemorrhages occur into the orbit blindness may result. A prolonged deficiency in vitamin C produces an enlargement of the suprarenal glands with consequent disturbance of their function. Among these disturbances may be those of vision and hearing.

The relation of vitamin D deficiency to the organs of special sense is most interesting. We have known for some time of the importance of this vitamin in prenatal life. We know that deficiency of this vitamin, like vitamin A, results in great muscular weakness. We conclude that deficiency of vitamin D during the later part of pregnancy, is often if not always, responsible for the various forms of squint that develop during early infancy. In these cases, where squint exists, there will be found other evidences of vitamin A and D deficiency.

All the mineral salts have not been studied with sufficient thoroughness to state in definite terms their influence upon the special sense organs. Therefore, I shall confine my remarks to a brief discussion of the relationship of calcium, phosphorus, iodine and iron to the special sense organs.

Calcium is a salt of tremendous importance in its influence upon the nervous system. We know that when it is reduced below 10 or 11 mg. per 100 cc. of blood serum we may expect serious nervous phenomena to appear. At the point of this reduction it is probable that Trousseau's, Chvostek's and Erb's signs may all be elicited and, when present, proves in the infant or child the condition known as tetany, or spasmophilia, and in the adult neuropathic phenomena. Since in these conditions we are able to trace a definite maternal transmission, we conclude that during pregnancy the mother suffered from calcium deficiency. We must also remember that the nervous manifestations of rickets—a disease in which calcium plays an important rôle—are general convulsions, laryngismus stridulus and tetany.

Were it not for calcium life would be in constant jeopardy from hemorrhage, because it is the chief factor in the coagulation of the blood.

Howell, Loeb and others have demonstrated that the heart muscle may be kept beating for a considerable length of time after removal from the body, when supplied, under proper conditions, with an artificial circulation of blood or lymph or water solution of blood ash.

Without a relatively large amount of phosphorus or one of its compounds, life cannot exist. Numerous experiments have proved that there can be no substitute for this element, as there may be for some of the other mineral salts. Unless it is sup-

plied in considerable quantities the more important organs, such as the brain and the special sense organs, cannot function. It is known that as the phosphorus supply is reduced there occurs a slowing down of all the higher functions.

The exact relation of blood phosphorus to adenoids has not been fully determined, but it has been observed that the blood phosphorus is much below normal in children when this defect exists.

Studies conducted within the past two years upon the subject of pernicious anemia, a disease heretofore thought to be intractable to treatment and always fatal, has greatly emphasized the importance of iron in the economy, and much light has been thrown upon the conditions under which iron is finally utilized in the tissues.

Investigations have proved that if liver pulp is daily fed to victims of pernicious anemia recovery will take place. However, sufficient emphasis has not been laid upon the kind of liver that is most effective. It is well known by investigators in the science of nutrition that calf's liver contains about five times as much iron as does the liver of the mature animal, and it is just about as good a source of the vitamins, provided the calf receives green food in its diet.

McCollum has expressed the opinion that the complete metabolism of iron depends upon its close association with vitamin E. We do not deny the importance of vitamin E in this connection, but we must not overlook the fact that the liver is the most important organ in the body for the storage of vitamin A, and both vitamin B and C are found in this organ in large quantities. Therefore until positive proof is presented to the contrary we feel safe in asserting that the close association of iron with vitamins A, B, C, D and E are the factors responsible for the complete utilization of iron.

Those of you who are familiar with pernicious anemia will at once realize the relationship between iron and the organs of special sense, because, as Sherman expresses it, iron stands in the closest possible relation to the fundamental processes of nutrition, being an essential element both of the oxygen carrying hemoglobin of the blood and of the chromatin substances, which appear to control (in some degree, at least) the most important and "vital" activities within the cells.

Iodin in its relation to the various forms of goiter and its close relation to the nervous system has occupied so much space in medical literature that we do not feel it necessary to make extended comment here. Thyroxin contains 65 per cent pure iodine. It is now accepted that the thyroxin, as discovered by Kendall, is essentially identical with the thyroid hormone as secreted by the gland, and that the thyroid utilizes inorganic iodine in the preparation of this hormone.

From the most recent studies we feel warranted in assuming that the influence of iodine extends to every organ in the body. In the disease which we know as thyrotoxicosis there seems to be a disturbance of the metabolism of iodine. There occurs an excessive secretion of thyroxin and consequently the system becomes poisoned. This disease is of infrequent occurrence in people who live along the seashore and who eat foods grown or produced in such sections, while it is of comparatively frequent occurrence in people who reside in the goiter belt.

It is not unusual to observe involvement of the special sense organs in such typical food deficiency diseases as pellagra, acrodynia, beri-beri, rickets, pernicious anemia and chlorosis. In pellagra inequalities of the pupils are very important diagnostic manifestations. When only one pupil is dilated it is almost invariably the right. Changes in the retina, anomalies of the fundus, especially the right, and atrophy of the optic nerve may all be observed in pellagra. In acrodynia there is a distressing photophobia present with lacrimation, also a perverted sense of taste and touch. We find in cases of long standing rickets frequent defects of vision and occasionally deafness. In chlorosis such manifestations as tinnitus aurium and black spots before the eyes.

In concluding my remarks upon this subject, may I remind you that while there has been great progress made in the elucidation of many of the problems of nutrition that seem mysterious, yet there are others that await explanation. Only recently one of them has presented itself to me in the form of a pellagrous mother, who brought her five-months-old baby to me suffering from acrodynia. The baby's sole source of nourishment was the mother's milk. The question naturally arises why should the same food deficiency react differently upon two individuals living under the same hygienic conditions

and the same environment and whose ultimate source of food was the same. To prove that the same deficiency existed, when the mother's diet was changed to one appropriate for the treatment of pellagra, both the mother and the baby promptly recovered.

May I ask you to be assured that while it is true that there are still many problems to be solved and no doubt will be just as successfully as has been the solution of pernicious anemia and many other equally important problems that for so long were considered hopeless, we do not have to wait for their solution because we know what foods are necessary to maintain normal health and well being. These are as follows: Milk, the leafy green vegetables (both raw and cooked), the whole grain cereals, the juice of citrus fruits, eggs, a moderate amount of meat, especially the internal organs, and certain of the tubers, such as carrots, white potatoes and yellow sweet potatoes.

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XLVIII.

THE PHYSIOLOGY OF THE LARYNX.

By W. V. MULLIN, M. D.,

CLEVELAND.

In presenting a discussion of the physiology of the larynx, I shall briefly consider three points of interest: (1) The reason for the midline position of the cord when the recurrent laryngeal nerve is injured; (2) adductor paralysis; (3) the causes of laryngeal paralysis; and shall add an interesting observation in regard to a malignant condition.

In thinking of the action of the muscles of the larynx, I like to compare it with the action of the muscles of the hand and fingers—by which the hand is both opened and closed. The action of closure is by far the more important function. When under normal conditions the nerves which control the muscles are stimulated, flexion takes place—that is, flexion or closure is the natural muscular function; while extension or dilatation, on the other hand, is a selective action which is wholly under the control of the will. From a functional standpoint, therefore, adduction must be regarded not merely as a movement of the cords toward the midline but as a closing up—a sphincter-like action of cartilages, ligaments and muscles alike. The normal muscular balance is such a perfect mechanism that it effects a complete coordination—such as will always exist unless this balance is destroyed. This coordination is made more evident by the conception of Negus that the vocal cords should be considered as valves which regulate the doorway to the thorax and control the functions of expiration and inspiration so as to assure the entrance and exit of the exact amount of air that is required for the performance of various specialized functions.

Congenital stridor in infants may be noted as an example of incoördination in the respiratory movements. This condition is well explained by Logan Turner, as due to a retarded development of the cortical centers which control respiration. A similar temporary condition occurs in children during anes-

thesia. According to Lack and Sutherland, likewise, the respiratory movements become incoördinated when the diaphragmatic intracostal respiratory movements are not synchronous with those of the abducting muscles of the larynx. Unilateral irritation of one or both the cortical centers causes complete bilateral adduction.

These observations agree with those made by Ballance and Colledge—that after anastomosis of the recurrent laryngeal nerve with the phrenic nerve, the tension of the paralyzed cord became more marked than that of the normal cord; that in six months the previously paralyzed cord moved more vigorously than the normal cord; and that seven months after the anastomosis the movement of the affected cord was almost violent in comparison with that of the cord on the opposite side. In experiments performed on animals it has also been shown that the movement of the cord could be increased by partial asphyxiation.

Abduction can be produced only by one muscle of the larynx—the crico-arytenoideus posticus muscle, the action of which is selective rather than natural. Because this muscle is large, it requires a greater nerve and blood supply than do the smaller muscles; therefore the function of this muscle can the more easily be impaired.

The cords of the larynx are rendered tense by the crico-thyroid muscle—which either pulls upward the anterior border of the cricoid cartilage, or pulls downward the anterior margin of the thyroid cartilage. This muscle is supplied by the superior laryngeal nerve.

The action of the principal adductor muscle, the crico-arytenoideus lateralis, is completed by the interarytenoideus muscle, which is supplied by one and sometimes two branches of the internal laryngeal nerve, a branch of the superior laryngeal nerve. This muscle is also supplied by the main part of the recurrent laryngeal nerve. Thus this single muscle receives a double nerve supply on each side. In this muscle, also, the branch from the recurrent laryngeal nerve is frequently joined to the branch from the internal nerve although neither branch crosses the midline.

These conclusions may be verified by those which were reached by Dilworth who referred to the laryngeal nerves as

"a highly modified plexus" which is produced by the separation of a strand of fibers from the vagus by the larynx, this strand becoming a continuous nerve which joins the internal and recurrent laryngeal nerves.

As yet I have been unable to find any explanation for the symmetrical position of the cords and the simultaneous recovery from bilateral interference with nerve function unless it be through influence from the higher centers.

2. Adductor Paralysis.—While I can accept this term as indicating "functional aphonia," clinically I cannot acknowledge such an entity as adductor paralysis, nor can I accept the idea that cold, syphilis, metallic poisoning, and like disturbances should affect only the adductor muscles. It is true that Saundby and Hewetson report a case which demonstrates an exception to Semon's law, but this case was one of an extensive carcinoma of the esophagus; therefore, there is a possibility that this condition caused degeneration in the muscles of the larynx. Personally I have not seen a case of true adductor paralysis.

3. The Cause of Laryngeal Paralysis.—I shall make no attempt to enumerate all the causes of laryngeal paralysis, but after making preoperative examinations of a large number of patients with goiter and making a careful study of our records for the past two years, I am prepared to say that enlargement of the thyroid gland is not a cause of laryngeal paralysis. We see an occasional case of laryngeal paralysis which is due to a malignant condition of the thyroid gland but practically never see such paralysis in patients who have non-malignant thyroid disturbances. In examining cases of laryngeal paralysis in patients who have enlarged thyroid glands, we find we have been able to locate the cause of the condition in the mediastinum. The recurrent laryngeal nerve is well surrounded by glands throughout its entire course. As these glands may readily become infected it would appear more plausible to suppose that pressure from these glands especially within the mediastinum is the cause of the laryngeal paralysis rather than the enlarged thyroid. In certain cases of bilateral paralysis the afferent and efferent nerve fibers of the vagus and of the superior laryngeal nerve are capable of transmitting the impulse through the bilateral center in the cortex, and even though

the stimulus, or injury, may affect the two sides unequally the effect on the movement and the position of the cords will probably be identical.

Jugular Foramen Syndrome, Right Side.—This condition is best described by the history of a typical case:

The patient was a man 38 years of age who on admission to the hospital complained of severe pain in the back about the lumbar region; of inability to swallow and of pain caused by a growth behind the angle of the jaw on the right side. The patient was markedly emaciated as a result of the lack of food and water.

On examination, there was found behind the angle of the jaw on the right side a hard glandular enlargement which overlaid the jugular foramen area. The patient was unable to open his mouth very far because of pain. There was no paralysis of the palatal muscles, but the tongue was slightly swollen and showed paralysis on the right side. The voice was slightly husky. The pharynx was filled with thick mucus—a condition usually seen in disturbances of the esophagus. Because of the large quantity of this mucus and because the patient was unable to open his mouth, it was almost impossible to see the larynx. In one fleeting glance, however, we found that the right cord showed no action but its exact position could not be determined. A feeding tube was inserted into the esophagus but the man lived only a short time.

At the postmortem examination the following findings were confirmed both grossly and microscopically: (1) carcinoma of the pancreas; (2) metastatic carcinoma of the liver, lungs, glands of the neck; and the mass extending into the jugular foramen on the right side was also a carcinoma. The larynx was removed in toto with the idea of determining definitely whether or not the right cord was paralyzed. This could not be determined from inspection as the larynx appeared normal in every way. A microscopical examination of the muscles, therefore, was necessary in order to determine whether or not degeneration had taken place. Much to our surprise this examination revealed the same microscopical picture of early carcinoma as was found in the microscopical examination of sections of the other organs.

CLEVELAND CLINIC.

XLIX.

CONSERVATIVE SURGICAL TREATMENT OF
CHRONIC MAXILLARY SINUSITIS.

BY JAMES C. TUCKER, M. D.,

BEATRICE, NEB.

According to Hajek, in the therapy of empyema of the maxillary sinuses, two main factors are to be considered:

1. Removal of the cause of the disease.
2. Free drainage of the pathologic secretion found in the sinus.

The exact cause of the disease is usually undetermined, hence therapy cannot be instituted on this basis in every case. The cases of dental origin are usually the easiest determined. Therapy based on the institution of drainage and aeration is the simplest type of conservative surgical treatment. We always give the conservative intranasal operation a trial before resorting to one of the so-called radical procedures. I think it is well that we should remember, when treating the nasal accessory sinuses, that we are dealing with disease of the respiratory tract. Also, I think it well to remember that most of the pathology of the nasal accessory sinuses is mucous membrane pathology. With these two ideas in mind, one will probably have more patience with and respect for conservative procedures.

A great many intranasal operations have been devised for the maxillary sinus. Krause was probably the first to advise operation beneath the inferior turbinate. I do not know who originated the operation we use. We call it the Mosher method. The inferior turbinate is turned up with a Luc forceps. This usually gives a fairly good view of the inferior meatus. Very rarely is it necessary to remove any of the anterior end of the inferior turbinate, or to even sever its anterior attachment. The mesoantral wall is punctured beneath the inferior turbinate with a frontal sinus rasp of the Gifford pattern. This opening is enlarged with a larger rasp of the Krause pattern. This opening is then enlarged with an ethmoid blade of the

Gruenwald-Struycken model. The lower part of the opening is made as near the nasal floor as possible. The Krause antral rasp or a Mosher ethmoid curette is very useful for smoothing down the lower edges near the nasal floor. We usually make an opening about one and one-half centimeters long by one centimeter up and down. In some cases it is not possible to get an opening of this size in the inferior meatus, in others it may be made longer if wanted. As a rule, no part of the inferior turbinate is removed. In some cases, where the inferior turbinate is markedly hypertrophied, it is resected beneath the mucoperiosteum and as much of the bone removed as required.

Septal deflections or other nasal pathology is usually corrected at the same sitting. In many cases multiple sinuses are opened at the same time. Where the septum is not resected, no packing is used unless to control bleeding. Bleeding is unusual. The after treatment consists in rest in bed for three or four days. The sinus is washed out on the third or fourth day following operation. They are given a vaselin ointment to use in the nose three or four times a day for a few days. The irrigations may be repeated if the discharge is profuse or if the sinus does not drain well. We are inclined to do less irrigating than formerly. Except in young children, local cocain anesthesia is preferred and advised.

We have records of 673 chronic maxillary sinus cases operated upon in the above manner. On five of this series a Caldwell-Luc operation or some other more radical procedure was done later. One case went elsewhere for a radical operation. Some of the others may have had treatment elsewhere, but we do not know of it.

Forty-seven of the above cases, or approximately 7 per cent, were of dental origin. In the majority of these the history indicated that the maxillary sinus symptoms followed dental surgery. We are rather inclined to follow the teaching of Dr. William L. Shearer, of Omaha, that maxillary sinus infection of dental origin more often follows dental surgery than otherwise.

In several cases in which polypous mucous membrane was seen in the sinus at operation or in which the radiographic report indicated polyposis within the sinus, the polyp delivered itself into the inferior meatus and was removed later.

In nine of the cases in which polypi could be seen in the sinus at the time of operation they could not be seen later.

A second operation was required in fourteen cases, as the original opening had closed or was not considered adequate for drainage and aeration. In four of the fourteen cases the sinus was reopened the third time.

Certain cases of maxillary sinus disease will require a radical operation, but most cases will improve under conservative intranasal procedures. Again I wish to state that in sinus disease the major part of the pathology lies in the mucous membrane, also that sinus disease is respiratory tract disease

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L.

A GROUP OF SINUS CASES PRESENTING INTERESTING CRANIAL SYMPTOMS.

BY EDWARD A. LOOPER, M. D.,

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The accessory nasal sinuses are anatomically so closely related to important areas of the brain, that it is not unusual to have severe cerebral manifestations develop from apparently trivial nasal infections.

There is a very intimate anastomosis between the blood supply of the sinuses and the cranial structures so that a small amount of toxin from a low grade sinus infection is sufficient to produce alarming mental symptoms if carried to some vital center.

During the past few years we have had the opportunity of seeing quite a number of such cases. Most of these patients have been sent into the hospital with suspected intracranial lesions, new growths or organic mental disturbances. But the most thorough examination failed to reveal any other cause for the trouble except a sinus infection, and after treatment of the sinuses, the symptoms cleared up.

Several of these patients were seen with Dr. Maurice Pincoffs of the Medical Department of the University of Maryland, and he has previously reported some of them in reference to the medical aspect, so I thought they might be of interest to you as laryngologists, to briefly present a few of the most interesting cases.

They are as follows:

Case 1.—J. B., a boy, 16 years of age, gave the following history:

Past History.—Tonsillitis at times; frequent colds. Past history otherwise negative.

Present Illness.—One week before entering the hospital, patient complained of a slight cold. He was put to bed but did

not improve. On the second day he complained of some frontal headache and a general feeling of malaise. On the third day, after attempting to get up, he fell back on the bed unconscious and expressionless. He slept continuously and could be aroused only with great difficulty, after which he would return to a semiconscious state. During the attack he vomited frequently and was unable to take food; he also had some photophobia. On admission to the hospital patient was still in a semiconscious condition, talked incoherently and with difficulty when aroused. Some intracranial condition was suspected and every possible investigation was made along that line. X-rays of cranium revealed no abnormalities. Pupils were dilated but round, equal in size and reacted slowly. Ocular muscular movements were normal. Vision could not be determined. Lens and media were clear. Optic nerves of normal color, outlines clearly defined. No evidence of choking or intracranial pressure. Other parts of eye grounds were normal.

Neurological examination showed that all reflexes were normal.

Mental test revealed that the patient was disoriented; hazy as to time; correct as to place; occasional slurring of words; concentration poorly sustained. On the second day in the hospital patient's mental condition was about the same. He developed a spasm of his left hand, which lasted a short time. He was still very dull, drowsy and apathetic. During this time a careful physical examination had been made, all structures appeared normal and no cause for the trouble could be determined. Dr. Pincoffs requested me to examine his nose and throat. I found a slight discharge in the right nostril, left antrum dark by transillumination and was cloudy on the X-ray plate. It did not seem to me likely that such severe mental symptoms should be caused by his nasal infection. However, as all other causes for his condition had been eliminated, we decided to make a simple puncture and wash out his antrum. This was done and a small amount of pus was evacuated. This contained staphylococci and a few streptococci organisms. Patient had no ill effects from the operation, and on the second day was very much improved. His mental condition was much clearer and he became rational. He gradually improved and at the end of four days was anxious to go home,

remembered a great deal about his illness and was mentally quite clear. He was discharged and has since reported a number of times over a period of about a year and has had no further symptoms.

Case 2.—M. C., a white boy, 18 years of age, gave the following history:

In July while riding in an auto, without warning, patient suddenly lost consciousness. This spell lasted for several minutes. Two weeks later, while getting up from dinner he had a similar spell. Thirteen days later, while riding in an auto he had his third attack. From this time on numerous spells developed which were associated with loss of consciousness, giddiness and falling. They frequently occurred when standing in one position or walking. The attacks lasted for several seconds and were usually followed by pain in the top of his head. Headaches increased in severity. A careful physical examination was essentially negative.

Patient was referred to me by Dr. Pincoffs for nasal examination and it was found that he had an infection of his left frontal sinus and ethmoids. By an intranasal operation his ethmoids and frontal sinus were drained, after which he had an uneventful recovery and has not had the slightest recurrence of his headaches nor mental symptoms since the operation.

Case 3.—C. C., white, 23 years of age, had the following notes made on his entrance to the hospital:

Patient, admitted on September 30, 1927, at 4 P. M., came stumbling into the accident room, complaining of poor vision and seeing double, also intense frontal headache. Present condition began about three weeks ago. He appears very nervous, answers questions slowly and is disoriented; can not remember dates, places or time. While in the hospital a very thorough study of his case was made, during which it was found that he had a great deal of infection of the right frontal sinus, ethmoids and left antrum with a mild infection on the left side. The sinuses were operated upon by me, after which he had a rapid recovery. He left the hospital apparently normal.

Case 4.—R. K., 50 years of age, gave an interesting history. He was a conductor on the B. & O. R. R., and had been discharged from service because he would frequently fall asleep

during his runs; also complained of attacks of vertigo at times and feeling confused. His mind was affected, concentration and memory disturbed and at times he was disoriented. A study of his case showed that his antra and ethmoids were badly infected. At the time of operation a great deal of thick foul pus was evacuated from both antra, after which patient's symptoms rapidly cleared up and in a few weeks he was able to return to his usual occupation, which he has steadily followed.

Case 5.—A man 37 years of age was sent to me from North Carolina with a diagnosis of suspected brain tumor. He was suffering with intense frontal headaches, limited to the left side and extending to the occipital region. He had a great deal of vertigo and nausea. When his pain and headache were most severe, there was a spasmodic twitching of the left arm, simulating attacks of Jacksonian epilepsy. He had photophobia and eyes would tire easily after reading. There was a history of repeated colds and obstruction to breathing on the left side. A general and special examination for intracranial new growths was negative. Examination of the nose revealed tenderness under the floor of left frontal sinus. The septum was deviated to the left. No nasal discharge. The left middle turbinate was very much hypertrophied. By transillumination, the left frontal sinus appeared darker than the right. X-ray showed a small left frontal sinus which was cloudy.

Patient was operated upon and in a week all of his symptoms had cleared up. He was free of the twitching in the left arm and has had no further trouble.

Case 6.—Mrs. E. G., 36 years of age, who was suspected of having a brain tumor on account of her violent headaches, nausea and vomiting, was referred to me for examination on June 5th, 1920. At that time she complained of having had attacks of severe frontal headaches on the right side of her head for a number of years. Recently the attacks have become very excruciating. She has extreme vertigo, at times unable to raise her head from the pillow. Nausea and vomiting most of the time. Patient was unable to think clearly and was not able to perform her usual work, memory poor and was very apathetic. Careful physical examination was made with particular attention directed towards the possibility of a new

growth, but the results were negative. Nasal examination revealed very definite infection of the right frontal sinus. She was operated upon, from which she had a gradual recovery, and in about a month's time was entirely free of pain, vertigo and mental symptoms.

Case 7.—F. G., aged 9, was admitted to the hospital with the following history:

On April 21st, patient developed a cold and tonsillitis. This was followed in three days by an erythematous rash over body, which lasted 24 hours. On April 28th, temperature went up to 104, she became semicomatose and remained in that condition until May 2nd, at which time an X-ray revealed empyema of the right antrum, ethmoids and frontal. The antrum was punctured and washed out, after which the temperature came down to normal and patient became rational. At 6 P. M., on May 2nd, the left arm and right side of face became partially paralyzed and at 5 P. M. on May 3rd she was seized with a convulsion. This was followed by two similar attacks very soon after.

Examination made by Dr. Pincoffs showed:

Onset of drowsiness, stiff neck and left facial weakness and weakness of left arm following acute tonsillitis and infection of sinuses. Two convulsions with improvement in the last few days. Temperature was up to 101-104 now down to 99-100.

Mentally clear but retarded. Speech slow, slight left facial weakness. Slight internal squint left eye. Tongue is midline. Loth to move head but it can be moved without marked pain. Tenderness over upper second cervical vertebrae. Definite weakness of left arm. All deep reflexes sluggish.

Impression.—“The case is entirely analogous to two similar ones seen with acute frontal sinusitis and brain symptoms which later cleared up completely. While there is a possibility of brain abscess I believe in this case, expectant therapy is the best while the case is improving.”

Diagnosis.—“Cerebral edema over right frontal sinus.” (Dr. Pincoffs.) At this time I was called in consultation to see the patient and found a definite infection of the right frontal sinus, ethmoids, sphenoid and antrum. This was also confirmed by X-ray. Nasal treatment was instituted and symptoms began

to improve and patient was able to leave the hospital in a short time. Reports from the patient four months later stated that she was entirely normal.

Case 8.—E. G., aged 25, consulted me on September 9, 1920, for sudden loss of vision in the left eye which had developed about a week previous to this visit. He had suffered no pain, gave no history of nasal infection or discharge. His complaint was that he arose one morning and found his vision very much blurred in the left eye, which continued to get worse until he could not see his hand. Associated with this was marked vertigo and slight nausea.

At the time of examination his left pupil was about 1 m. m. larger than the right. Muscular movements were normal in all directions. Vision was limited, for he could make out only the tips of my fingers at one foot. Visual field, as shown on chart, was concentrically contracted to ten degrees. He could not distinguish colors. Ophthalmoscopic examination showed slight hyperemia and faint blurring of the edges of the optic disc. Fundus otherwise negative.

The extraocular structures of the right eye were negative. Pupillary reactions good. Ocular movements normal. Vision 20/20. No pathologic changes made out in fundus. Visual field normal.

Nasal examination showed marked clouding of left antrum by transillumination and confirmed by X-ray examination. There was no tenderness over the sinuses. No nasal discharge. Breathing space unobstructed. In fact, if transillumination and X-ray had not been used, there would have been no findings to indicate sinus involvement.

On September 11, 1920, the left antrum was drained and a small quantity of seropurulent discharge evacuated. The following morning when I visited him in the hospital, to my surprise he exclaimed, "I can see perfectly well," and on testing his vision I found that he could read fine newspaper print without difficulty. He did not complain of headache or vertigo, and felt fine.

The next day on taking his visual field I found it had improved about 15 degrees, and in five days had returned to

normal with normal vision. The hyperemia of the optic disc had also disappeared and there was no vertigo.

Case 9.—J. D., aged 41, colored. On November 14, 1926, patient was walking down the street, had a convulsion, fell unconscious on the street, attack lasting for about 10 minutes. This convulsion consisted of twitching of all muscles bilaterally. He was frothing from the mouth and injured his tongue and lip during the convulsion. Patient was brought to the accident room of the University Hospital and during admission had another attack which was a fifth, and while in the hospital had several similar attacks. Nothing in the patient's physical examination could be found to explain his condition, except a possible antral infection, as both were densely cloudy. After irrigation of the antra a great deal of pus was evacuated. Patient's mental conditions gradually cleared up and he was discharged on November 30th, 1926, at which time all of his symptoms had disappeared.

Case 10. M. N., aged 12 years, was admitted to the hospital with the following history:

Three days before admission, patient complained of some headache, nausea, pain in abdomen. She vomited after eating, was put to bed feeling very drowsy but responded to questions. The next day she became semidelirious, temperature 104, vomiting increased. Diplopia and delirious most of the time.

Tentative Diagnosis.—Encephalitis and Lethargica. Examination of sinuses showed emphyema of left antrum. This antrum was punctured and washed out. Patient's symptoms immediately began to clear up, and in about two weeks' time she was entirely well.

Comment (by Dr. Pincoffs).—"I should class this case with a number of others we have had of a temporary palsies, aphasias, convulsions, etc., associated with acute sinusitis and clearing up spontaneously. Such symptoms must be due to localized cerebral edema."

SUMMARY.

These cases have been presented because they have had such a common group of mental symptoms, the severity of which appeared out of all proportion to the amount of sinus involve-

ment. Most of the cases clearing up after a simple antral puncture and drainage.

The most probable explanation would seem to be a cerebral edema due to the reaction of toxins, carried directly to the brain by the close relationship of the vascular anastomosis.

It also demonstrates the importance of making careful routine nasal examinations in all medical cases, and particularly in suspected cases of encephalitis, brain tumors, epilepsy or unusual cerebral manifestations.

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CYTOLOGIC EXAMINATION IN THE DIAGNOSIS OF INFECTION IN THE NASAL ACCESSORY SINUSES.*

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While I feel that this method may develop its usefulness in all the nasal accessory cavities, I have used it only in the maxillary and sphenoid sinuses. It has furnished me most assistance in the maxillary sinus investigations, and I shall therefore confine my remarks in this preliminary article to that sinus.

We depend upon many things for our diagnosis of maxillary sinusitis. Among the important factors to be considered are (1) history, (2) symptoms, (3) physical findings, (4) X-ray examination, (5) cytologic examination, (6) irrigation. I wish to discuss only the last two methods mentioned.

It has been a common practice for years to enter the antrum with a trocar of some sort and irrigate it, washing the contents of the antrum with the wash water out through the nose into a basin where it is collected for study. Of all the methods of diagnosis this has been considered the most reliable. Hajek, for instance, in his excellent book but recently off the press, goes so far as to say: "If the irrigating fluid returns clear, no mucus or purulent secretion is present in the antrum."

This method, I have become convinced, is open to definite objections. It is not trustworthy. Where there is enough discharge in the antrum of a certain type it may be conclusive. However, it has been shown that it often gives no indication of the true antrum content. The following simple facts from a case observed by me show the inadequacy of this crude

*After the proof of this article was sent to the publisher, the author found a reference to the work of Doctor Watson-Williams, *Journal of Laryngology*, 1912. Accordingly he wishes to accord full priority to Doctor Watson-Williams for his pioneer work in this field.

method.¹ The X-ray in this case showed both antra definitely diseased, with polypoid thickening of the mucous membrane. A No. 16 needle was introduced in the usual manner into the right antrum. A 5 cc. Luer syringe was fitted direct and aspiration performed. Air only was withdrawn, and this suggested that the antrum was empty. Sterile water was injected through and caught in a basin as it emerged from the nose. It was put into a glass and held before the light for observation and was absolutely clear. The same type of needle was inserted into the other, or left antrum, and upon withdrawing the piston, the 5 cc. syringe filled up with an opaque, viscid material. It was about the color of ground glass. This known, viscid material was next dropped into a glass of sterile water and it disappeared instantly. I later operated upon the antra and found the mucous membrane thick and polypoid in both, as shown by the X-ray in the first place. We, therefore, know that the pathologic material accumulated in an antrum may be completely soluble in water and, therefore, not recognizable by the ordinary methods of antrum irrigation.

For years some men have insisted on using a black catch basin when washing out the antrum, contending that they picked up disease that would be overlooked in a white basin. Just as the black basin is better than the white, so is the microscope or chemical reagent an improvement upon the naked eye.

The ordinarily used method of irrigation is not only in error from the standpoint that it overlooks disease, but is objectionable because the return flow out through the nose may carry material lodged there from other sources.

It has been the practice of a few men for some years to inject sterile solutions into the antrum and withdraw and examine them bacteriologically and, I think, chemically. These methods are excellent and productive of great help but require expert assistance to be of any reliability, and even then it is very difficult to be sure of lack of contamination in the manipulation. After a number of years of trial, these methods have not come into popular use.

The method here described is simple and can be carried out in the course of office practice. It is based upon the fact that mononuclear and polymorphonuclear leucocytes are never pres-

ent in any appreciable numbers in healthy accessory sinuses.

Although it may open up considerable variance of opinion and lead the reader away from the definite scope of the paper, I cannot refrain from adding that mononuclear and polymorphonuclear leucocytes are normally not only foreign to the sinuses, but that they are also absent, in any numbers, in the mouth, nasopharynx and nose of absolutely healthy individuals. There are other factors than sinus disease to produce these cells in the mouth and nasopharynx and possibly in the nose. As examples I mention pyorrhea, diseased tonsils, diseased adenoids, foreign body or atrophic rhinitis, etc.

Slides made successively from secretion from the mouth, pharynx, nasopharynx, nose, and lastly the sinus, have led us as surely to the source of infection as the grains of gold found in the gravel of the creek bed have led the prospector upward, pan by pan, to the mother lode.

Applied to the antrum our method is as follows: The nose is cocainized underneath the inferior turbinate. A sterile No. 16 needle, without handle or obturator, is introduced into the antrum in the usual manner. Care is taken not to pass the needle against the opposite antral wall because it may cause bleeding. Blood, while confusing, is rarely encountered and can be distinguished under the microscope. When the needle is in place a 5 cc. Luer syringe is connected directly and the piston is withdrawn. If fluid appears, it is placed in a sterile tube for examination. If, however, air is drawn freely into the syringe we may know that the point of the needle is free in the antrum and that the opening into the nose is patulous.

Twenty cc. of sterile water or isotonic solution is warmed to a pleasant temperature in a small 40 cc. Florence flask. The 40 cc. size will admit the barrel of a 5 cc. Luer syringe. The head of the patient is bent well over toward the corresponding side, in the position best favoring retention of the fluid in the antrum. The syringe is connected again and the warm solution is injected slowly into the antrum. The patient is asked to tell if the solution is felt to go down the throat. After 5 or 10 cc., more or less, according to the free available space in the antrum, have been injected, the piston is slowly withdrawn and the recovered fluid is placed in a sterile test tube for examination. Both sides are usually studied, where nec-

essary, at the same time, a separate flask of solution and another syringe, of course, being used.

Where the fluid returned is contaminated sufficiently, a slide may be made directly from it. However, where there is little or no evidence in the returned solution, it is put into a centrifuge and a slide made from whatever may be deposited at the point of the centrifuge tube.

Normal antra show no cellular content whatever; low grade infection is productive of mononuclear leucocytes; while the more active processes furnish either polymorphonuclear leucocytes alone, or polymorphonuclear mixed with mononuclear cells. Or in the same antrum we may find pus lying on the floor containing only the polymorphonuclear type of cell, while above the pus the cavity of the antrum may furnish a preponderance of round cells in the injected and aspirated fluid.

This interesting observation was brought to my attention by Mrs. Hooe, my technician, in studying the following case.

The patient complained of nasal discharge, yellow in color. The X-ray pictures showed a fluid level in the right antrum with mucous membrane thickening; mucous membrane thickening only in the left antrum. A needle was introduced into the right antrum, aspiration performed, air only filling the syringe. It was evident that the point of the needle was above the level of the pus. Sterile fluid was now injected and aspirated. The return fluid was centrifugalized and showed a preponderance of round cells. The needle was now made to engage the pus. This was very thick and only a little could be withdrawn into the syringe. This returned mucopus showed itself to contain only polymorphonuclear leucocytes. Fluid was then injected through the antrum and collected in a dish as it came from the nose. The wash water was clear, but a yellow mass appeared at the anterior naris. This was pulled out with forceps and consisted of a mass of yellow, thick mucopus. The observation of the two kinds of cells, practically unmixed, found in the same antrum, seems to be of considerable importance. The mucopus itself was composed mostly of polymorphonuclear leucocytes. It was thick and kept its place in the lower part of the antrum when the patient was upright, but was affected by gravity on change of position of the head. The remaining part of the antral cavity had recovered from

the more acute inflammation and furnished only round cells. These round cells were probably washed from the walls of the sinus by the injected solution which even came in contact with the thick mucopus. The mucilaginous character of the mucopus held the polymorphonuclear leucocytes from mixing with the injected solution.

This method has taught us that many antra are diseased that we formerly have considered normal. Many interesting facts have been elicited that have given us new ideas as to diseased antra. Where the antra have been operated upon, the mucous membrane is examined histologically, and this check has been invaluable in estimating pathology. We find that the X-ray is of the greatest aid in diagnosis, and we apologize for the heated arguments formerly had with the X-ray department when we got a report of "great mucous membrane thickening in the antrum," and washed through and saw nothing in the wash water. Except in the cystic variety we rarely find true mucous membrane thickening without finding the explanation for it in the cytologic study.

Cystic degeneration of the mucous membrane is an interesting pathologic condition. The cyst may fill the antrum completely and, if so, the needle introduced taps the fluid and it runs out freely like fluid from an ascitic abdomen. Cysts of large size are often shown in the X-ray plates, rising up free in the antrum. It has been possible to pass the needle into the antrum missing the cyst. Injection and withdrawal of the solution has shown accompanying disease when present. Then the needle has been made to engage the cyst, rupture it, returning the typical yellow fluid characteristic of cyst content. We have introduced the needle into the antrum, withdrawn the piston, getting nothing but air. Then, on filling the antrum with our solution and withdrawing again, we have gotten thick, yellow mucopus in quantity. The needle evidently passed above the level of the pus.*

It is also common to find it impossible to withdraw the piston after connecting with the needle. This is due to one of three things: (1) The needle has become stopped up. If that is the case the sterile wire obturator is passed through the needle,

*Observation of my associate, Dr. J. A. Bacher.

removing the obstruction; (2) the point of the needle is engaged in thickened mucous membrane or possibly thick, inspissated discharge. It may be freed in some cases by moving the point of the needle about until an open space is found. In some instances the antrum may be so filled up with polypoid tissue or thickened secretion that no aspiration is possible. In such cases the X-ray will show the antrum entirely opaque, although it may not be able to distinguish in such opacity between mucous membrane thickening and fluid. The following case illustrates the limitations of the needle puncture as a diagnostic measure. In this patient the X-ray showed the left antrum entirely opaque. It was impossible to tell whether there was mucous membrane thickening. The needle was easily introduced into the antrum, but no aspiration or movement of the piston of the attached syringe was possible. At operation the antrum was found entirely filled with inspissated pus. It had almost the consistency of heavy automobile grease. When placed in alcohol it closely resembled polypoid mucous membrane. The mucous membrane in this antrum was hardly thickened, although a little edematous.

3. The opening into the nose has become stopped up from thickening of the mucous membrane in the sinus or in the nose. Sometimes there is a valvelike action which permits air or fluid to pass in one direction but not in the other.

Great care is taken in all these manipulations not to inject any air until we are sure of the position of the needle because of the danger of air embolism.² We are usually able to aspirate air, which decides that the needle is free in the antrum.

The cytologic study of antrum content is especially useful in considering those cases with repeated exacerbations of antrum infection. Low grade inflammation, as shown by mononuclear cell content, is frequently present continuously between the acute attacks.

The following cases are reported very briefly. An analysis of them brings out some facts and leads to speculation.

It should be made plain that operative indications are not to be based necessarily upon the findings of mono- or polymorphonuclear leucocytes in the sinus. This data is added to the other clinical factors that determine the judgment of the surgeon.

Operations, where performed, were done under local anesthesia, using a modified Caldwell-Luc technic. The mucoperiosteum was entirely removed and was examined histologically in the surgical laboratory by Professor F. E. Blaisdell, or my first assistant, Dr. Leland Hunnicutt. Working through the antrum, an incision through the fontanelle just above the inferior turbinate was made with a knife, the nose entered and the lateral wall of the middle meatus largely removed by ring punch forceps. No flap of mucous membrane from the nasal mucosa was retained for lining the antrum.

Cases Nos. 100, 1574, 886, 1545, 101, 1660, 1234, 1614, 102 and 1760 show polymorphonuclear leucocytosis in the cytologic examination and chronic sinusitis in the pathologic report.

Cases Nos. 1625 and 1614 (left side) show mononuclear leucocytosis in the cytologic examination and chronic sinusitis in the pathologic report.

Cases Nos. 100, 1574, 1545, 1234, 1614 (right), 102, 1760, 1666 (right), 1858, 1807, 1806, 103 and 1240 show pathology in the X-ray plates and polymorphonuclear leucocytes in the cytologic examination.

Cases Nos. 1685, 1614, 1810 and 1858 (left) show pathology in the X-ray plates and mononuclear leucocytes in the cytologic examination.

Cases Nos. 886, 101 and 1660 show negative X-ray findings and polymorphonuclear leucocytosis in the cytologic examination and chronic sinusitis in the pathologic report. These cases show that we cannot always rely upon the X-ray plates alone.

Case No. 1737 shows pathology in the X-ray plates, polymorphonuclear leucocytes in the cytologic examination made at the time, and mononuclear leucocytes when examined two weeks later. This case shows the change in the character of the cytology that takes place very commonly as the acute stage subsides.

Cases Nos. 1666 (left), 1591, 496 (left), 1805 and 1876 (left) show pathology in the X-ray, and polymorphonuclear leucocytes in the cytologic examination. Patients were not operated upon.

Cases Nos. 104, 1579, 1805 (left) and 1757 (right) show pathology in the X-ray plates and mononuclear leucocytes in the cytologic examination.

Cases Nos. 496 (right), 1757 (left) and 1876 (right) show no pathology in the X-ray but polymorphonuclear leucocytosis in the cytologic examination.

Case No. 105 shows negative X-ray plates and mononuclear leucocytes in the cytologic examination.

Cases Nos. 886, 101, 1660, 1625, 1614 (left), 102, 1760, 1810, 1858, 1806, 103, 1240, 1591, 1805, 1737 and 105 show polymorphonuclear leucocytes in the cytologic examination, where the returned water was almost clear, and would probably have passed for normal without the aid of the microscope.

Mrs. M. (100).—Symptoms: Headache off and on for many years.

History: Frequent colds. Excess nasal and postnasal discharge.

Physical findings: Some mucopus seen in nose at times during observation, and samples sent in show very profuse, yellow discharge from nose.

X-ray: The maxillary sinuses are completely filled, presumably with fluid.

Cytologic examination: Pus aspirated from both antra. Slides show numerous pus cells, polymorphonuclears predominating.

Irrigation: Large quantity of pus washed from both sides.

Operation: Double radical antrum, January 29, 1927. Local anesthesia. Very polypoid mucous membrane and much pus, both sides.

Pathologic report: Sinusitis, catarrhal chronic.

Mrs. L. W. MacD. (1574).—Symptom: Cough, chronic "catarrh," bronchitis.

History: Excess yellow discharge from nose and nasopharynx, many years' duration.

Physical findings: Right side of nose suggestive of sinus infection. Left side, no discharge is seen. Tremendous amount of yellow discharge sent in on samples.

X-ray: Both maxillary sinuses are filled, presumably with fluid.

Cytologic examination: Small amount of yellow pus aspirated from right side. Slide shows numerous pus cells with polymorphonuclears predominating. Slide, left side, shows scattered pus cells, polymorphonuclears predominating.

Irrigation: Considerable pus washed from right side.

Operation: Double radical antrum, December 14, 1926. Local anesthesia. Mucous membrane greatly thickened and polypoid in character.

Pathologic report: Sinusitis, chronic catarrhal, most marked on left side.

Miss B. P. (886).—Symptoms: Asthma, many years duration.

History: Excess yellow discharge from nose and nasopharynx.

Physical findings: Very little discharge seen in the nose, but brings in samples with quantities of discharge on them, definite, yellow.

X-rays: Nothing noted in the maxillaries.

Cytologic examination: Sterile water into both antra. Very few small flakes in water returned. Slide shows on the right side scattered polymorphonuclears. Slide shows on the left side considerable number of polymorphonuclears and a few mononuclears.

Irrigation: Water looks clear. Very few small flakes of mucus seen.

Operation: Double radical antrum, November 12, 1926. Local anesthesia. Distinct yellow pus in right antrum, mucous membrane thickened. Left side, mucous membrane not so thick and polypoid as on right.

Pathologic report: Right, sinusitis, chronic infective; left, sinusitis, chronic.

Miss R. C. (1545).—Symptoms: Very frequent "colds."

History: "Colds" four or five times a year, with yellow discharge from nose.

Physical findings: No discharge seen in the nose, but sends in samples showing definite yellow discharge from the nose and nasopharynx.

X-rays: Right maxillary filled, presumably with fluid.

Cytologic examination: Needle into right antrum, bright yellow cystic fluid poured out through needle, about 5 cc. Slide shows a predominance of endothelial cells with a few small lymphocytes and occasional polymorphonuclears.

Operation: Right radical antrum, December 21, 1926. Local anesthesia. Mucosa quite thick in most parts, in other places slightly polypoid.

Pathologic report: Sinusitis, chronic.

Miss M. E. (101).—Symptoms: Gastrointestinal disturbance, headaches.

History: Runs low grade temperature. Feels bad all the time. No appetite, loses weight. Some yellow discharge from the nose at times, feels better when the nose discharges.

Physical findings: Not much discharge seen in the nose, but brings thick, yellow discharge on samples.

X-ray: Possibly some trouble in right ethmoids, but nothing noted in antra.

Cytologic examination: Sterile water injected into both antra and aspirated. Water returned fairly clear from both sides. Slides show numerous mononuclears and a few polymorphonuclears on both sides.

Irrigation: Water returned fairly clear.

Operation: Double radical antrum, December 21, 1926. Local anesthesia. Mucosa quite thick in most parts, in other places slightly polypoid, less on left.

Pathologic report: Sinusitis, subacute catarrhal.

Mr. R. M. (1660).—Symptoms: "Cold" for past month with cough and bronchitis.

History: Has had a cold for a month with postnasal dropping.

Physical findings: Some mucopus seen in both sides of the nose, more on the right side.

X-rays: "Normal sinuses."

Cytologic examination: Sterile water into right antrum and withdrawn, few shreds of mucus in returned water. Left side, few flakes mucus in water returned. Slides show, on the right side, considerable number of cells with a predominance of polymorphonuclears. Left side, quite a large number of cells with a predominance of polymorphonuclears.

Irrigation: Right side washed, water returned with a few shreds of mucus. Left side washed, thick, yellow pus in water.

X-rays, second report: After sinuses had been washed and pus found in left side, had more X-rays taken. Report: "Greater density where water has been left indicates that the

maxillaries did actually contain air before. There must have been some secretion distributed about the surface of the mucous membrane not sufficiently regular to give sufficient outline but enough to make them appear very slightly gray."

Operation: Double radical antrum, December 7, 1926. Local anesthesia. Thick, yellow pus on right. Thick yellow pus in anterior part on left, cyst in posterior part.

Pathologic report: Sinusitis, chronic catarrhal, double.

Mrs. F. S. H. (1234).—Symptoms: Diabetes, hay fever and sneezing.

History: Has been a semi-invalid for a number of years due to diabetes. Says there is no yellow discharge from her nose.

Physical findings: Polypi seen in both sides of nose removed.

X-rays: Left maxillary half full of fluid. Heavy mucous membrane thickening on right.

Cytologic examination: Thick, yellow pus aspirated from both antra. Slide shows numerous mononuclears and polymorphonuclears, about evenly divided, on the right side. Slide from left side shows many pus cells, polymorphonuclears predominating.

Operation: Double radical antrum, December 3, 1926. Local anesthesia. Mucous membrane thickened and polypoid. December 18, mononuclear leucocytosis, W. B. C. 1350, polys 1 per cent, monos. 96 per cent. December 19, W. B. C. 5000, polys 59 per cent, monos. 37 per cent. This patient is well. Low sugar content unimportant.

Mrs. M. F. (1625).—Symptoms: Frequent sore throat and "colds," bad headaches.

History: History of frequent "colds" with excess, yellow nasal discharge.

Physical findings: Little mucus seen in both sides of nose. Samples from nasopharynx. Definite evidence of sinus disease.

X-rays: Both maxillaries are filled up, presumably with fluid.

Cytologic examination: Sterile water injected into both antra and aspirated, water returned clear. Slide from right shows a few mononuclears; from left numerous mononuclears.

Operation: Double radical antrum, November 30, 1926. Local anesthesia. On the right side, a thin layer of pus curetted

from the mucosa. Left side is the same except that the mucous membrane was more polypoid and there was more pus present.

Pathologic report: Sinusitis, catarrhal infective.

Mrs. F. P. W. (1614).—Symptoms: Severe arthritis.

History: Pain in right shoulder and arm, quite severe at times. Duration, three years. Excess yellow discharge from nose and nasopharynx, worse the past year. Tonsils removed two and one-half years ago—no relief.

Physical findings: No discharge seen in the nose. Samples of discharge sent in with profuse yellow discharge showing.

X-rays: Right maxillary completely filled, presumably with fluid. Mucous membrane thickening on left side.

Cytologic examination: Sterile water injected into right antrum returned cloudy, but no definite pus seen; left side returned clear. Slides show numerous mononuclears on both sides, and a few polymorphonuclears on right.

Operation: Double radical antrum, November 12, 1926. Local anesthesia. Mucous membrane edematous—looked as if antra were filled with serous fluid. Scraped left side slightly with curette before removing mucous membrane and filled curette with yellow pus.

Pathologic report: Right, sinusitis, chronic catarrhal, polypoid. Left side, sinusitis, chronic catarrhal, more polypoid than on right.

Miss G. T. (102).—Symptoms: Deafness, several years' duration.

History: Formerly asthma, none since coming to California. Excess, yellow discharge from nose.

Physical findings: No discharge seen in nose, but sends in samples of definite yellow nasal discharge.

X-ray: Polyp in right antrum.

Cytologic examination: Sterile water injected into right antrum; very few shreds of mucus in water returned. Slide shows quite numerous mononuclears and scattered polymorphonuclears. Sterile water into left antrum, water returned clear. Slide negative. January 27, 1927. Radical right antrum. Very polypoid mucous membrane.

Pathologic findings: Sinusitis (antritis), catarrhal (polypoid, edematous).

Mr. A. K. (1760).—Symptoms: Tinnitus, some deafness.

History: Tinnitus has come on gradually past seven or eight months. Polypi removed from nose many times. Asthma as a child and frequent colds. Frequent headaches and extremely nervous.

Physical findings: No discharge seen in nose. Suspicion of a polyp seen in right posterior naris.

X-rays: Either polyp or cyst in right antrum. Left antrum obscured, probably by fluid.

Cytologic: Needle into right antrum; first could not withdraw piston of syringe, moved needle and withdrew air. Sterile water injected and withdrew a few flakes; then on movement of the point of needle, yellow, foaming fluid ran out. The needle had engaged the cyst. Slide shows numerous mononuclears and a few polymorphonuclears. Needle into left antrum, but could not withdraw piston of syringe even after searching about with needle point.

Operation: Double radical antrum, February 22, 1927. Local anesthesia. Definitely thickened lining on right side. On the left side, the antrum was entirely filled with a tenacious pus, which, upon removal, showed a clean, thickened mucosa. The inspissated pus resembled polypi when removed, as it was in one piece. Resembled automobile grease in consistency.

Mrs. E. D. (1810).—Symptoms: Five attacks of erysipelas since September, 1926.

History: Trouble with nose for four years. Pimples form in vestibule and also further in. Condition has now become chronic. Chronic inflammatory condition at tip of nose. One side of nose always blocked. Excess postnasal discharge.

Physical findings: Turbinates engorged on both sides of nose, purulent crusts on both sides, very definite evidence of sinus disease.

X-rays (not taken at Stanford): "Clouding of left antrum."

Cytologic examination: Sterile water injected into left antrum, agitated and withdrawn, returned clear. Slide shows considerable number of mononuclears.

Irrigation: Wash water appeared clear, but few small flakes of mucus seen on holding to light.

Operation, February 21, 1927: Radical antrum, left. Local anesthesia. Slightly thickened mucosa.

Pathologic report: Sinusitis (antritis), subacute catarrhal.

Mr. Y. F. (1666).—Symptoms: Noise in right ear.

History: Discharge from nose, summer time clear, winter time greenish yellow. These symptoms present several years. Headaches, formerly more severe. Deafness in right ear.

Physical findings: Some nasal and postnasal discharge seen.

X-rays: Suspect some mucous membrane thickening on floor of right maxillary.

Cytologic examination: Sterile water injected into right antrum, agitated, withdrawn; some mucus seen in the water. Slide shows thick clusters of mononuclears and polymorphonuclears. Same procedure on the left side. Slide shows clusters of mononuclears.

Operation: Double radical antrum, March 7, 1927. Local anesthesia. Slightly thickened lining on both sides. Smear made at time of operation and examined under microscope shows on the right, clusters of round cells; left side, not quite so many round cells and an occasional polymorphonuclear.

Mr. L. W. MacD. (1858).—Symptoms: Yellow, purulent discharge from nose.

History: Subject to colds. Profuse yellow discharge from nose, more from the right side.

Physical findings: Nose, right side, mucous membrane inflamed and bathed in pus. Middle turbinate is swollen. Left side looks better. Large amount of thick, yellow pus seen coming down on posterior pharyngeal wall. Profuse yellow discharge on samples brought in.

X-rays: Right maxillary sinus completely filled. Dense mucous membrane thickening of the lining membrane of the left maxillary.

Cytologic examination: Needle into right antrum and pus aspirated. Slide shows great numbers of polymorphonuclears and mononuclears.

CASES OBSERVED BUT NOT OPERATED UPON.

Dr. E. C. (103).—Symptom: Hay fever.

History: Excess yellow discharge from nose, sneezing, frequent colds. Submucous resection in 1923.

Physical findings: Mucopurulent discharge seen in the nose at times during observation. Some yellow discharge brought in on samples.

X-ray, January 28, 1926: There has appeared a thick pad of mucous membrane hypertrophy in the lower outer portion of the left maxillary. February 22, 1926. Heavy pads of thickened mucous membrane remain in the left maxillary. The right maxillary now presents the same appearance.

Cytologic examination: Sterile water injected into left antrum. Many small shreds seen in return fluid. Slide shows numerous polymorphonuclears and mononuclears.

Irrigation: Water returned very shreddy. Was very painful.

Mrs. G. (1240).—Symptom: "Weakness." Excess nasal discharge.

History: Better since tonsillectomy one year ago but not well. Frequent "colds," attacks of sneezing. Lots of discharge from nose, more from the right side.

X-ray: Mucous membrane thickening in both maxillaries. There is some fluid level in each, which shifts when the patient changes her position.

Cytologic examination: Needle into right antrum, aspirated about 5 cc. almost clear, watery fluid. Slide shows considerable number polymorphonuclears. Water into left antrum and returned slightly gray. Slide shows a few polymorphonuclears.

Irrigated: Wash water somewhat shreddy, both sides.

Mr. H. J. (1591).—Symptom: Bronchitis.

History: Frequent "colds," almost constant since "flu" in 1919. Excess postnasal discharge but none anteriorly.

Physical findings: No pus seen. Space is good. Sends in very small amount nasal discharge.

X-rays: Negative.

Cytologic examination: Sterile water injected into right antrum and aspirated returned clear. Slide shows numerous polymorphonuclears.

Mr. E. D. (104).—Symptoms: Chronic sinusitis, many years' duration.

History: Frequent "colds" with yellow discharge from nose.

Physical findings: Mucopus seen in the nose at times during observation. Definite yellow discharge sent in on cloth.

X-rays: Little mucous membrane thickening in both maxillaries.

Cytologic examination: Right antrum entered with needle but could not withdraw piston of syringe. Needle into left antrum and grayish material aspirated. Slide shows numerous mononuclears.

Colonel W. (1658).—Symptoms: Cough and discharge of quantities of pus from lungs, 35 years' duration. Negative for tuberculosis.

History: Frequent "colds," discharge from the nose and nasopharynx, generally yellow.

Physical findings: No discharge seen in the nose. Samples of discharge brought in show definite yellow discharge from both sides of the nose.

X-ray: "There is rather marked cloudiness of all the accessory sinuses. I believe all of them contain an extensive amount of granulation tissue and probably some pus."

Cytologic examination: Aspirated right antrum, water returned somewhat bloody. Slide shows scattered pus cells, polymorphonuclears predominating. Sterile water into left antrum but could not aspirate very well, as piston of syringe could not be withdrawn.

Mrs. H. (1579).—Symptoms: Loss of vision.

History: Discharge from nose constantly and frequent colds.

Physical findings: No discharge seen in the nose but sends in samples of definite yellow discharge.

X-rays: Polyp in right antrum. Slight inflammation left antrum.

Cytologic examination: Sterile water injected and withdrawn from both antra. Returned very slightly cloudy, possibly more so on right. Slides show scattered mononuclears on both sides.

Irrigation: Wash water slightly cloudy.

Miss C. I. (496).—Symptoms: Dull headache, stiff neck, atrophic rhinitis.

History: Has had a scintillating scotoma for years—three attacks in the past six weeks. Atrophic rhinitis, several years' duration.

Physical findings: Sends in samples of definite, yellow discharge from nose.

X-rays: Some mucous membrane thickening left maxillary.

Cytologic examination: Sterile water injected into right antrum returned somewhat cloudy. Slide shows clusters of mononuclears. Sterile water into left antrum and aspirated. Water returned cloudy. Slide shows numerous mononuclears and a few polymorphonuclears.

Irrigation: Thick pus washed through on left side. Small amount of mucopus washed through on right.

Miss M. M. (1805).—Symptom: Swelling over left antrum, eight days' duration.

History: Had an acute head infection three weeks ago. Has a lot of nasal discharge, but less the last few days. Excess postnasal discharge for a long time.

Physical findings: Swelling over left antrum slightly tender. Turbinates engorged on both sides. Mucopus seen in nasopharynx.

X-rays: Left maxillary contains free fluid which has a shifting level. Heavy mucous membrane thickening in right maxillary.

Cytologic examination: Sterile water injected into the left antrum and withdrawn, water practically clear. Slide shows cluster of round cells. Unable to force wash water through. Two days later, needle again into left antrum. Could get neither air nor water through at first, then, on manipulation of the point of the needle, seemed as if something suddenly broke and very thick mucopus aspirated. Slide shows numerous leucocytes, both mononuclears and polymorphonuclears.

Irrigation: No pus apparent in wash water, only few small flakes of mucus.

Mr. F. B. (1737).—Symptoms: Heavy, dull feeling in head, unable to concentrate.

History: Had a cold for ten days. Three days ago profuse yellow discharge from nose, more from the left side. Has had more or less trouble of this kind for past two years.

Physical findings: No discharge seen in either side of the nose or nasopharynx.

X-rays: Left maxillary completely filled. Much density in posterior ethmoids.

Cytologic examination: Aspirated thick pus from left antrum. Patient became faint, so did not irrigate. Month later,

needle into left antrum, sterile water injected and aspirated, few small flakes of mucus in water. Slide shows numerous round cells.

Irrigation: Wash water looks clear but few flakes of mucus seen on holding to light. Symptoms still present.

Mrs. J. J. M. (1757).—Symptoms: Discharge from the nose, four years' duration.

History: Discharge is yellow when she has a cold and watery the rest of the time. Some rheumatism occasionally.

Physical findings: Considerable hypertrophy of middle turbinate, mucous membrane suggestive of sinus infection.

X-rays: Little mucous membrane thickening about the right maxillary.

Cytologic examination: Needle into right antrum. Had considerable difficulty in getting air through. Sterile water injected and withdrawn. Water quite bloody but appears to be some flakes of mucus in it. Slide shows scattered mononuclears. Sterile water injected into left antrum and withdrawn, cloudy. Slide shows many polymorphonuclears and many mononuclears.

Mr. W. S.—Symptoms: Some chest pathology, heart lesion.

History: Nose always stopped up in the morning. Some postnasal discharge. Myocarditis. Tonsils removed; symptoms continue.

Physical findings: No definite discharge seen in the nose. Sends in quantity of definite, yellow discharge on muslin.

X-rays: The sinuses appear clear.

Cytologic examination: Needle into both antra, sterile water injected and withdrawn. Water clear except for few small flakes. Slide shows clusters of round cells on both sides, less on the right.

Dr. E. D. (1876).—Symptoms: Hay fever and asthma.

History: Yellow discharge from both sides of nose for years. Postnasal dropping, more or less bronchitis.

Physical findings: Quantity of polypoid tissue seen on the right, crowded under middle turbinate. Left side looks about the same. Brings samples of discharge from nose and nasopharynx, definite yellow discharge from right side of nose and nasopharynx. He usually has more discharge from the left side.

X-ray: "Clouding of ethmoids. Some symptoms pointing to left antrum."

Cytologic examination: Sterile water injected into right antrum and withdrawn; some flakes of mucopus in water. Water went in all right, but very hard to withdraw piston of syringe to aspirate. Slide shows clusters of round cells and an occasional polymorphonuclear. Sterile water injected into left antrum, hard to aspirate; flakes of mucus in water returned. Slide shows clusters of round cells, occasional polymorphonuclear, less cells than on right side. Slide made from material in nasopharynx; scattered pus cells, with polymorphonuclears predominating.

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LII.

QUALITY HEARING.*

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The present study was undertaken to determine standards or criteria of performance in various groups of persons who were to be regarded as having hearing powers above the average, either as the result of superior health or through a specialized training influencing aural acuity; further, all members of the several groups were selected on the basis of absence of any demonstrable aural disease or impairment. The real goal was the standardization of a technic of observation, which would permit the determination of significant relative acuity measurements and quality interpretations under the usual conditions of clinical practice. In the collation of large amounts of data, care must be exercised both in recording and in interpreting. The average is but a mathematical quantity defining a linear relationship. The normal, on the other hand, is the individual whose departure from the average assumes insignificant proportions.

Movement and sensation are the two extremes of the processes of human life by which the individual is brought into direct relation with the outer world. Movements are always objective in character and can be studied directly by external observation. Sensations are invariably subjective, and can be directly analyzed only by introspection; they are indirectly inferred from the expressional movements which are their external concomitants. Thus the messages from the different organs to the centers may definitely cross the threshold of consciousness and give rise to distinct sensations which differ in quality and intensity. But very few of the impressions that

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reach us from the outer world, or from our own body, enter completely into consciousness, because the attention can be focused upon only a small part of the impressions received.

It is important to notice that two kinds of dissimilarity can be distinguished in the comparative study of sensations. No one, for instance, can say whether a given musical tone resembles more closely the color red, or a bitter taste. If, on the other hand, we compare the sensations appreciable within each modality, we can certainly recognize qualitative differences. Thus two separate auditory sensations may be qualitatively distinguished by their difference of pitch; it is also possible to judge which of them is the stronger or more intense. The colors of the spectrum present not only a gradual transition from one to the other, but we can also appreciate their greater or lesser resemblance to each other or their relative brightness. The different modalities of the sensations do not depend on differences in the external stimuli which excite them, but on the specific nature of the different senses.

The instruments used in this series of observations were the audiometers No. 1-A and No. 2-A (Bell Telephone Laboratories, Inc.),* which is essentially a vacuum tube oscillator, equipped with the necessary controls for regulating the frequency and the output. The oscillator generates single frequency oscillating electric currents which produce tones in the specially designed receiver. By means of the controls, tones of any one of the several pitches at octave or half octave intervals may be produced, and the intensity of these tones may be varied. The difference between the two audiometers is that No. 1-A is the larger, runs on a storage battery, and is designed to fulfill the requirements for an extensive examination of the acuity and the quality of hearing. Its pitch range, divided into twenty stops, extends from 32 to 16,384 double vibrations per second, and the controllable variation of its intensity covers approximately the entire hearing range.

With the audiometers in question, the pure tones of the oscillator are transmitted to the ear through a specially de-

*Note.—The authors take the greatest pleasure in acknowledging their indebtedness to Dr. D. L. Lynch, also to the New England Telephone and Telegraph Company, through which company the machines were made available for this and other studies.

signed microphone. Pressures can be varied at will by the operator, and the threshold of pressure perception of the subject determined for each frequency. In general, the technic followed in making these observations and measurements was, with but few exceptions, that advised by Bell Telephone Laboratories, Inc. The most significant precautions are:

1. The performance of the tests was in a nearly noiseless room, as all the selected subjects were as near the normal in hearing as it was possible to obtain, and thus slight deviations were most significant.

2. The patients were allowed to become familiar with the machine and its several different tones before the records were taken.

3. The dial was rotated anticlockwise until the tone had entirely ceased, the reading of the stationary calibrated scale being noted. Next, the dial was rotated clockwise until the sound was again perceived and the dial reading again recorded. By successive rotations the threshold point can be fairly evaluated. An interrupter button permits a variation of the procedure, and, in conjunction with the resistance, permits the elimination of certain misleading elements arising equally from lack or excess of cooperation. The acuity curves were obtained with the No. 2-A machine.

4. The foregoing method may be repeated as often as necessary to convince the operator of the correctness of his observations. Care should be taken, however, not to repeat the tests too many times, as the auditory nerve is readily fatigued.

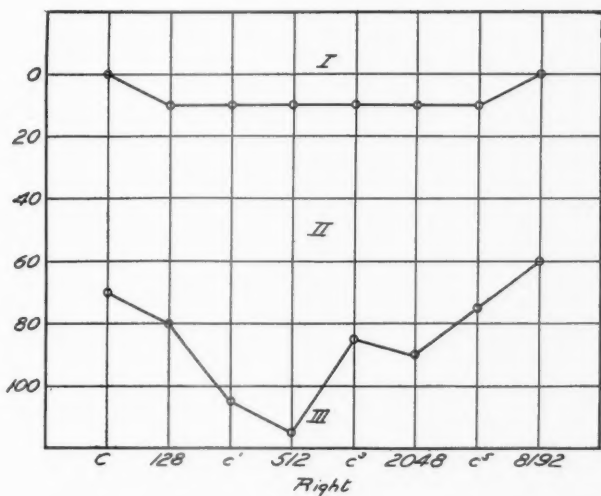
5. To determine the quality of the tones the large machine was used, and the words recommended were before the patient throughout the measurements. The shadings between the meanings of the many words found in the several columns seemed rather confusing to a number of the subjects. The quality curves were determined with the No. 1-A instrument.

In recording the results, the conventional diagram suggested by Fletcher has been used throughout. The dial of the instruments are calibrated to indicate the hearing loss in sensation units, and readings are transcribed directly on the chart. The change in quality of the tones was also transcribed directly on the chart. By a further convention, the observed hearing loss and the changes in the quality of tone perception

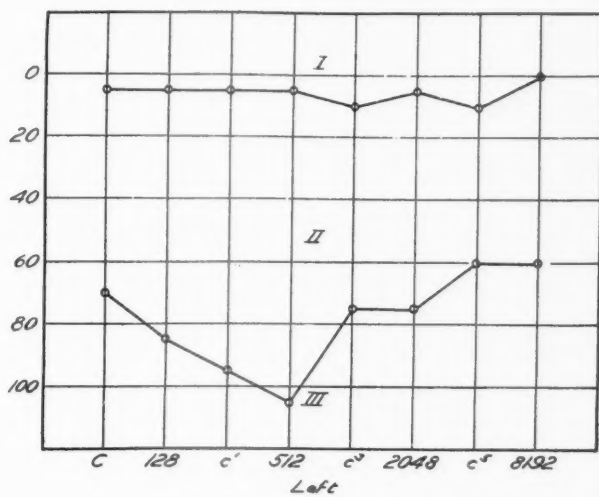
may be designated in terms of percentage, this representing the relative position of a point observed between the base line of normal hearing and the curve of total loss.

A total of 70 subjects have been examined in this study, of whom 46 were males and 24 were females. That the work as compiled might be thoroughly representative of acuteness of hearing, seven groups of ten subjects each were selected. There were in the various groups ten telegraphers, ten telephone operators, ten marine radio operators, ten expert piano tuners, ten ornithologists and twenty college athletes, ten boys and ten girls used as controls. Further, as the study is preliminary in character, young trained subjects were selected, as the failure of normal hearing quality incident to advancing years would introduce an extraneous factor of variation and thus somewhat influence the significance of the averages.

A characteristic plot for the right and left ear for one group is illustrated below. These plots show just below the zero line the acuity curve. The area (I), which extends from the acuity curve upwards to the zero line, is that of no sound for the individual or group. Below the acuity curve, and extending over the major portion of the graph, is the second area (II), or area A, which includes the entire ability of the individual to perceive the several musical tones through the several frequencies. Area (III) extends from the lower limit of area A to the limits of the machine. It apparently is the pathologic area, or the point wherein the musical tones cease to exist and a disagreeable noise begins. This point is found to be very constant with the individual each time the test is applied.



Graph 1.



Graph 2.

The combined data of the several averages may be presented most compactly in tabular form and are collected in Tables I and II. For the sake of ready comparison, the areas are expressed in percentages. Thus at C, 64 d. v., the male athletes have 9 per cent of the range in the soundless area, 89 per cent in musical tone and 2 per cent in the noise area. Table I represents the two control groups, while Table II pictures the remaining fifty subjects.

TABLE I.

Frequency	Areas	Athlete Male	Athlete Female
	1	9	8
C = 64	2	89	63
	3	2	29
	1	5	7
c = 128	2	82	71
	3	13	22
	1	6	8
c ¹ = 256	2	83	78
	3	11	14
	1	6	7
c ² = 512	2	81	74
	3	13	19
	1	5	6
c ³ = 1024	2	82	73
	3	13	21
	1	4	5
c ⁴ = 2048	2	79	68
	3	17	27
	1	5	4
c ⁵ = 4096	2	70	65
	3	25	31
	1	1	2
c ⁶ = 8192	2	63	59
	3	36	39
	1	5	6
Average	2	80	69
	3	15	25

TABLE II.

Frequency	Area	Telegrapher	Telephone Operator	Radio Operator	Piano Tuner	Ornithologist
C = 64	1	16	20	19	25	19
	2	82	80	79	67	63
	3	2	0	2	8	18
c = 128	1	10	15	10	14	12
	2	83	72	78	69*	69
	3	7	13	12	17	19
c ¹ = 256	1	10	14	9	11	11
	2	85	73	72	71	75
	3	5	13	19	18	14
c ² = 512	1	9	15	8	11	9
	2	82	62	72	66	68
	3	9	23	20	23	23
c ³ = 1024	1	9	13	7	7	8
	2	83	67	71	68	73
	3	8	20	22	25	19
c ⁴ = 2048	1	8	12	7	5	8
	2	78	66	56	64	62
	3	14	22	37	31	30
c ⁵ = 4096	1	11	12	5	4	8
	2	66	58	63	60	61
	3	23	30	32	36	31
c ⁶ = 8192	1	10	7	9	10	7
	2	55	57	43	49	58
	3	35	36	48	41	35
Average	1	10	14	9	11	10
	2	77	67	67	64	66
	3	13	19	24	25	24

With the exception of the male athletes and the telegraphers, the middle speech area is nearly a third larger, while the rest of the groups are about 25 per cent only. It will be noted that seldom are the two ears of the same person exactly alike in sensitiveness, although showing no differences objectively. This applies primarily to the acuity curve, as in the quality analysis the two ears seem to approach much nearer each other in response. The point of change from the normal to the pathologic area is very sharply defined and remains very clear cut. On the other hand, the acuity of an ear will vary from time to time, or even in the same day as much as ten sensation units. In the vast majority of the cases the nor-

mal quality area was uniform and extended throughout all the intensities. If one ear showed quality changes, apparently the other ear would show about the same change and at about the same intensity.

The significant point to be drawn from both the acuity curve and the quality curve is the obvious influence of physical well being and nutritional level on aural interpretation. While the acuity curve shows a uniform tendency toward slightly lessened acuity in the low and improved perception, in the high frequencies the quality curve shows a decided uniformity above the upper limits of the speech center and practically no pathologic spots at the upper frequency. Knowing that the difficulties in testing hearing are greater than are those in testing sight, none the less, it is felt that the acuity curve together with the quality curve forms a distinct design or pattern, and the combination may be an aid in differentiating disease conditions of the intracranial pathways. It is to determine this point that the study is ultimately directed.

The observations on 16,384 d. v. are collected together in Table III. The data here are recorded in sensation units. They can be readily transformed into percentage of hearing loss. Each number is the average for each individual in each group.

TABLE III.

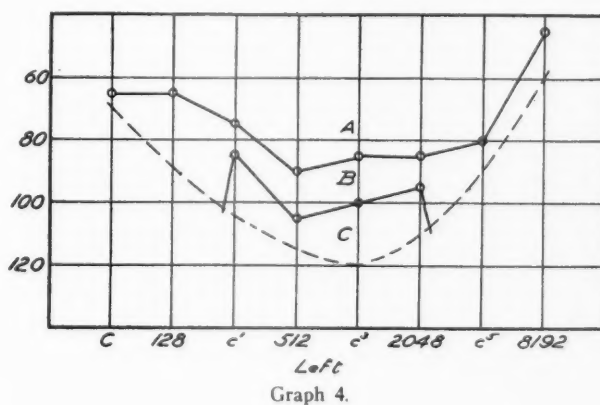
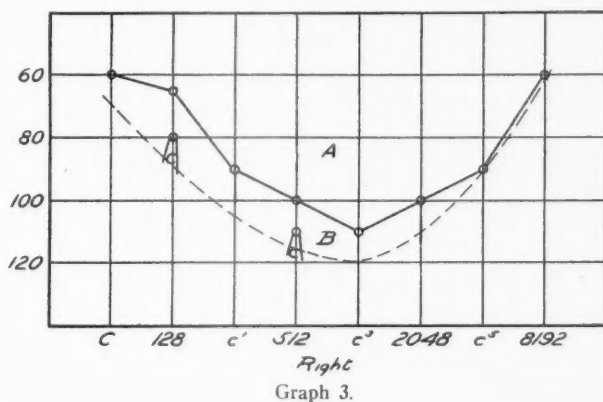
Case No.	Ear	Athlete Male	Athlete Female	Telegrapher	Telephone Operator	Radio Operator	Piano Tuner	Ornithologist
1	R	5	—	10	—	15	—	—
	L	15	10	—	0	0	5	—
2	R	—	5	—	10	—	10	—
	L	—	5	—	15	—	5	—
3	R	10	10	10	—	—5	—5	10
	L	15	10	10	0	5	—5	—
4	R	5	15	15	15	—	15	—
	L	10	—	15	—	—	15	—
5	R	—	—	—	5	5	15	—
	L	5	10	—	0	10	—5	—
6	R	20	10	—	—	0	10	—
	L	20	5	—	—	10	15	—
7	R	—	15	—	10	5	20	—
	L	10	20	—	10	20	15	—
8	R	15	10	—	—5	15	15	—
	L	5	10	—	—5	10	10	—
9	R	10	15	—	—5	20	—	—
	L	10	15	—	5	—	—	—
10	R	10	15	—	0	—	10	—
	L	10	15	—	0	—	—	—

It will be noted that about 70 per cent heard distinctly in one or both ears the highest frequency. The control group of girls all heard the tone, while only one ornithologist was recorded. Many of the various groups mentioned the fact that that frequency would not have registered in their consciousness unless they had been on the lookout for it and were being tested especially for acuity. An interesting fact was that not one member of all the groups could determine any shade of quality to the frequency. The tone was lost very quickly indeed, even when the intensity was increased. This may be due to a more rapid fatigue element entering into the picture. Another factor entering into the problem may be that the lower tones mask the higher, even when the high tones have greater intensity. There was practically no difference between the two ears.

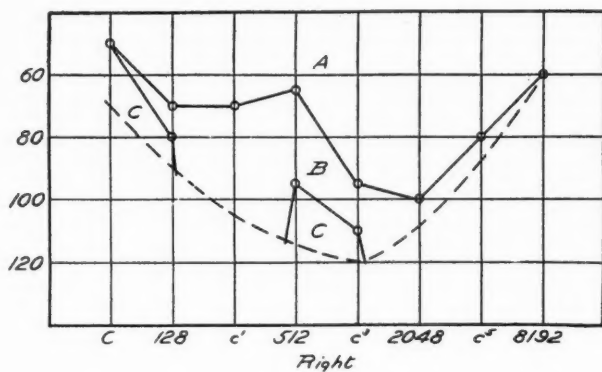
It appears that about 10 per cent of the total study have no A-area, or ability of quality differentiation. The anomalies occurred most frequently on the 64 d. v. frequency, showing one ornithologist deficient in both the right and left ear, one telegrapher in the right ear and three of the female controls that were quality deaf in both ears at the lower frequency. The telegrapher mentioned the fact that it made no difference with his ability to listen to and read the key, as the sound that he listened to all day was a mechanical noise and not a musical tone. The sequence showed one piano tuner quality deficient to each frequency in the left ear. As he is unique in this regard in the total group of 70, it is a warrantable inference that he possessed an aberration of hearing which disqualified him for this study. He mentioned the fact that he always closed the left ear with cotton when testing the tone of the various strings. Several of the radio operators showed loss of quality hearing at individual frequencies. They were the only group to exhibit this anomaly. Further, they were the only members that showed any quality deafness on the 8192 d. v. frequency. The boy athletes and the trained telephone operators all had quality hearing on each frequency at all intensities. Several of the radio operators distinguished two tones at the same intensity at the upper edge of the speech area. They were able to concentrate on one or the other quality at will. The telegraphers were the most consistent of all

the different groups in the regularity of the A-area. Several of the men could listen to a message from the box at the right ear and carry on an accurate telephone conversation with the left ear while typing the wire message. None of the above cases showed any middle ear anomalies, and each member showed a rather high acuity curve.

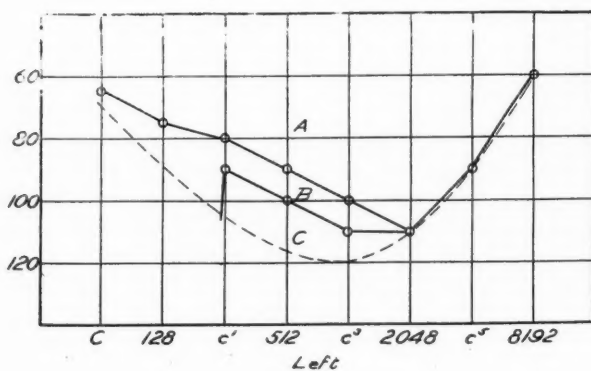
In the following graphs of the right and left quality fields it is noticeable on the first two (piano tuner), the increased A-area in the right ear with the small C-areas and the lack of quality hearing at the high frequency, while in the left ear C-area is larger, as is B-area, with B-area extending over a lessened part at the lower frequencies.



In the next two graphs, which are from a trained telephone operator, the right ear shows a smaller A-area in the center, but her acuity curve at this point was less good than at the other frequencies. Both ears show restricted abnormal areas in the upper frequencies, which is characteristic in 90 per cent of the group.

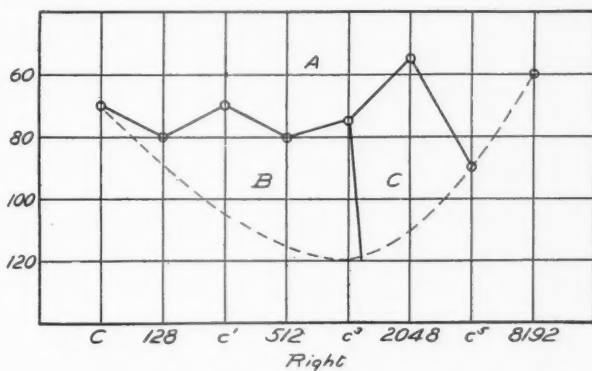


Graph 5.

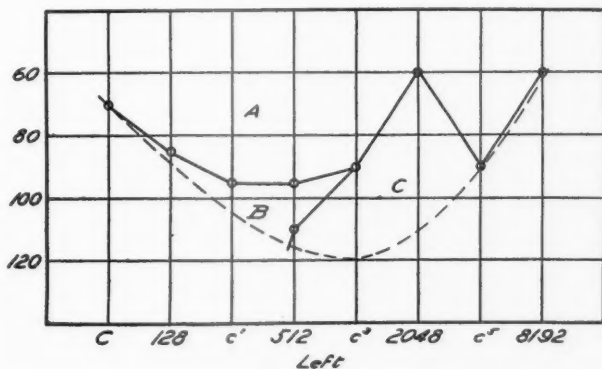


Graph 6.

The last two graphs are from a marine radio operator. As a group they show no B and C-areas on the high frequency. With one exception their A-area is extensive, and only the exceptional man showed a B or C-area and then on the upper edge of the area of speech.



Graph 7.



Graph 8.

These several graphs are typical of the group that they represent. I offer the suggestion of a likeness to the eye in that there may be an analogy between the acuity curve and

the mechanism of eye accommodation. On the other hand, delineation of the quality curve may be considered as simulating the plotting of eyegrounds.

The boundaries of the transition regions are usually more sharply defined than those of lesser audibility, and the sensations in these areas are so radically different from the sensation of a pure tone that it is with difficulty that the subject is convinced that the stimulation is the same pure tone to which he has been listening at the other intensities. Several of the subjects seemed to think that something radically wrong had happened to the machine and, on opening their eyes, would ask that the tone be tried again that they might convince themselves of its correctness. The regular sequence of B and C-areas following A is very striking. In no instance has the process been reversed.

CONCLUSIONS.

1. The determination of differences in quality hearing in these individuals of superior acuity and training offers the possibility of significant and characteristic limitations in certain disease conditions.

2. Some hearing mechanisms that are seemingly normal still have very small abnormal quality areas.

3. Very pure tones are essential in making the tests.

4. A certain number of people with normal acuity have no powers of determining quality differences and are unaware of their limitations.

In conclusion, the author wishes to express his sincerest indebtedness to Dr. Allan Winter Rowe, and to thank Dr. Reginald Hunt for his assistance with the graphs.

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LIII.

PULMONARY HEMORRHAGE IN CASES OF PULMONARY TUBERCULOSIS, CHRONIC BRONCHIECTASIS AND MITRAL STENOSIS.*

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In reviewing the older literature on pulmonary disease, one is impressed with the significance accorded pulmonary hemorrhage in the diagnosis of pulmonary tuberculosis. Although this disease was not always diagnosed on the basis of pulmonary hemorrhage alone, such bleeding was usually attributed to a tuberculous focus until proved otherwise. Later, when the roentgen ray was used to aid in the examination of the lungs, it was observed that in many cases of pulmonary hemorrhage there was no demonstrable pulmonary lesion. It was, therefore, assumed in such cases that the hemorrhage was from a tuberculous focus near the hilum of the lung.

This conception of pulmonary hemorrhage prevailed until further aid in the diagnosis of pulmonary disease was afforded by bronchoscopy. Direct inspection of the trachea and bronchi revealed that many lesions other than those of tuberculosis cause bleeding from the lungs. Benign and malignant neoplasms, pulmonary abscess and foreign bodies are some of the less common lesions giving rise to pulmonary hemorrhage. More common causes are chronic bronchiectasis and chronic mitral endocarditis with stenosis.

The records of 100 patients suffering from active pulmonary tuberculosis have been reviewed and compared with those of an equal number of patients suffering from chronic bronchiectasis and also an equal number suffering from mitral stenosis, with dyspnea on exertion, in order to determine the relative incidence of pulmonary hemorrhage in these diseases. In none

*Read before the American Bronchoscopic Society at Atlantic City May 21, 1927.

of the cases was there a lesion other than the essential one to account for the bleeding.

Twenty-nine of the group of patients suffering from active pulmonary tuberculosis had had pulmonary bleeding during the course of the disease. In eleven cases the hemorrhages had been slight, in fifteen moderate, and in three severe. In none was the hemorrhage very severe. Hemorrhage did not follow exertion in any of the cases. There were multiple hemorrhages in eleven cases. It is of interest to note in connection with this small series that Heise, at the Trudeau Sanatorium, has observed pulmonary hemorrhage in 34 per cent of 1,062 cases of pulmonary tuberculosis previous to the patients' admission to the sanatorium.

In cases of chronic bronchiectasis is seen the highest percentage of pulmonary hemorrhage. It had occurred in forty-nine of this group of cases. In twenty cases the hemorrhage was slight, in fifteen moderate, in nine severe, and in five very severe. In twenty-four cases the hemorrhages were multiple. Hemorrhages followed exertion in four cases, in two of which it was precipitated at any time by undue physical strain.

Eighteen of the group of patients suffering from mitral stenosis, with dyspnea on exertion, had had pulmonary hemorrhage. The hemorrhage was described as slight, or "streaks of blood only" in ten cases. In five cases it was moderate, in two it was severe, and in one very severe. Several hemorrhages occurred in the same patient in twelve cases. Hemorrhage followed exertion in seven cases.

While it is true that pulmonary tuberculosis is usually the cause of pulmonary hemorrhage, because of the greater frequency of the disease, yet its relative incidence is much greater in cases of chronic bronchiectasis, and the hemorrhage is more severe, more frequent and more often related to exertion.

Although pulmonary hemorrhage is absolutely and relatively less common in mitral stenosis than in pulmonary tuberculosis, it is common enough to warrant consideration in differential diagnosis.

LIV.

A DISCUSSION OF SOME OF THE CLINICAL
PROBLEMS OF CHRONIC SINUSITIS.

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POCATELLO, IDAHO

In the diagnosis and treatment of chronic nasal accessory sinus infection, there is a great deal of difference between adults and children. I shall restrict this discussion to the former only.

A large part of the work of a rhinologist is seeking for hidden foci of infection. I cannot imagine a field where the problems are more exacting. If the general surgeon finds the infection in the appendix and he removes it, the trouble is entirely taken away and the patient recovers. If he finds it in the gall bladder, the same result can be obtained. The same thing holds true when the foci are found in any of the accessible parts. They can be freely incised. They can be curretted. They can be cauterized. The different medicinal and mechanotherapeutic agents can be freely used and the pathologic processes wiped out. They do not return because they are completely removed. The parts then heal, the tissues return to normal and function returns as before they were invaded. This is not true of infection in the paranasal sinuses.

Given a definite infection in one sinus, there is always very great danger of adjoining ones becoming involved. It is not new to you when I say that the outlet of one sinus is the outlet of many sinuses. The blood supply may be the same and certainly the lymph channels draining the infected ones are the same as drain the normal ones. We well know how infection can spread in contiguous territory if disturbed by trauma, either by the surgeon's knife or by topical applications carelessly applied. Then our problem is this: we must remove the infection from the paranasal cells without spreading it to others not yet involved, and in so doing we must conserve all the normal nasal tissue that is compatible with good ventilation and drainage. We must relieve the patient of his

symptoms and restore him to good general health with a nasal mucous membrane that will function without discomfort.

I have repeatedly noticed that the postoperative reaction of patients, to a marked extent, depends on what part of the nasal chamber has been operated upon. We may injure the inferior turbinate with the actual cautery. We may incise it with knife or scissors, removing it in part or the whole. We may infract it sharply and then take down the nasointral wall in the inferior meatus, and the patient will have but very little systemic reaction. We can remove spurs from the septum in the region of the floor, and again there is very little disturbance, either general or local. On the other hand, just as soon as we do a submucous resection on the septum, or remove the middle turbinate, any or all of it, the postoperative period is marked by more pronounced symptoms. The patient's head will ache, his eyes will get red and watery, his face will flush, his nasal passages will close from swelling, and quantities of serosanguineous discharge will pass from his nose. His ears will feel full and his head will ring. He will often vomit, become constipated and his appetite will be poor. It is not unusual for the patient to be extremely nervous and restless. Often a sedative is required to give him rest and sleep, and when up and about, after two or three days, he is pale and weak. He has the appearance of one who has been ill.

Recently theories have been advanced that the glands in this area are related to those of internal secretion. The conclusion is justified by marked improvement in ethmoiditis, hay fever and kindred affections, when the extract of some of these glands is administered. We all know that adrenalin has a place in the treatment of these diseases, and I have seen some very favorable results from the judicious use of thyroid extract.

When we meddle in the olfactory fissure or the mucous membrane covering the middle meatus, the middle turbinate, the superior turbinate, the upper septum and the nasal side of the ethmoid and sphenoid sinuses, we are in supersensitive tissue. A fine delicate touch with a complete knowledge of the anatomic relations are needed.

I am confronted every day by this symptom complex. Patients come complaining of frequent colds, or a constant cold,

or an inability to withstand the slightest exposure without contracting a "fresh" one. They have irregular, indefinite headache. At times this is characterized by what the patients term a "fullness" in the head. They frequently say that if something could only be put high in the nose, so that it would open up, they are sure relief could be obtained; and this in patients who have no obstruction in the air passages whatsoever. At times the pain will be across the brow or definitely through the bridge of the nose, but also extending back "under the eyes." At times the pain is definitely located over one frontal sinus, thus giving all the classic signs of an infection in this area.

When more carefully observed, both clinically and by the skiagraph, the frontal will be found to be normal. The symptoms are probably referred. These are the vacuum frontal sinus headaches described by Sluder.¹ The infundibulum closes by swelling of the ethmoids and the middle turbinate, the air is absorbed and negative pressure causes the pain. The nose is dry part of the time, and part of the time there is more nasal secretion than normal. If the patient is recovering from an acute exacerbation, the secretion is peculiar in that it stains the handkerchief lemon color and is more mucoid than purulent. The middle turbinates, in these cases, are enlarged. They fill the vault by crowding the septum and narrow the middle meatus. Accompanying these symptoms is a general ill feeling, a capricious appetite, constipation, general nervousness, restlessness at night, awaking in the morning unrefreshed. Pallor is another symptom. This seems to be vasomotor, since it is present when the patient feels badly and is gone on those days when he feels improved. The red count is near normal and the hemoglobin well up. The tongue shows teeth indentations and is coated. At times there is an ischemia about the vermilion lip borders.

These individuals can well be called "nervous, run-down patients." I have treated them with daily topical applications in the office, shrinking down the tissues with the hope that I would promote better drainage. I have used Dowling's² argyrol tampons and autogenous vaccines after the method of Dr. Harold Hays.³ I have used diathermy and many other remedies, all with varying success. One author in the New York

issue of the Clinics of North America, October number, 1925, gives an excellent account of patients with these very symptoms, but he attributes it, not to the local condition, but to one of auto-intoxication from the intestines, particularly the large one. He reports excellent results by putting them on a carbohydrate free diet and regulating the bowels by ichthyol enemata, plain oil and Pillsbury's Health Bran. I have had no experience with this method, but it sounds logical and is worthy of a trial. We all know that diet has something to do with nasal infections. Fasting causes freer breathing, while after a full meal the nasal mucous membrane becomes congested. I am not so enthusiastic about the idea of the local condition causing all the trouble, for it may well work the other way and some other disturbance may cause the ethmoiditis.

The purulent type is very different. It is always an interesting differential diagnosis to determine whether the pus is coming from the ethmoid labyrinth or from the maxillary sinus, even though the X-ray shows the corresponding antrum somewhat blurred. I have often observed cases where pus constantly poured into the middle meatus and where polyps formed in this area, which when treated by conservative methods, such as the simple removal of the polyp, cleaning out the middle meatus and a few office treatments, rapidly recovered.

On the other hand, the great majority of these cases are far more resistant to treatment. I believe that when a chronic suppurative ethmoiditis is present, palliative treatment should be used only for a short time. Even though the condition improves at first, it usually recurs with the next head cold. When this does occur, it is best to open the entire sphenoid labyrinth. I believe with Yankauer⁴ that when part of the ethmoids are opened, eventually all have to be opened. These patients will return with the same symptoms, or with symptoms even more marked, and the complete operation has to be done later. I have had better results when the middle turbinates are left in place. I am firmly convinced that this highly specialized structure should be preserved when not too badly involved in some pathologic process, or when by enlargement of a narrow bone, it will not permit of drainage. I will quote from Pratt⁵, "Nearly all discussions on ethmoid

work bring forward the abnormalities found in the ethmoid cells, but they are few and far between. You may find them possibly in 5% of the cases, and it is seldom that they cannot be reached under the middle turbinate. When you do an exenteration under the middle turbinate, you have perfect drainage with no loss of physiology, and if it is done properly, you make this group of cells into one large cell, into which opens the frontal sinus. Any abnormally situated cell can be traced by the discharge and the enlarged ostia. Of course, there are causes that necessitate external operations, but they are rare."

If the middle turbinate is completely removed and all the ethmoid cells cleaned out, as well as the anterior wall of the sphenoid taken down, there is too much scar tissue formation to allow the nose to function properly. The function of ciliated epithelium cannot be taken by scar tissue. In health these delicate cells constantly keep wiping the mucoid secretions backward and do not permit of dryness and scabbing. Again, with the ostium of the maxillary sinus wide open, there is more danger of an infection from above. I have observed this several times in my own practice as well in that of others. It is always disconcerting to do a proper spheno-ethmoid operation, only to be compelled in a few months to open the antrum and treat this large sinus, recently infected. Therefore, I believe that unless there is a definite indication for its removal, the middle turbinate should be left in place.

I am convinced that the sphenoid sinus is more perplexing than any of the others; first, because of its inaccessibility to clinical observation; second, because our X-ray technic has not yet been perfected enough to aid us in the diagnosis as it does in others, particularly the maxillary and frontal, and third, because of its close proximity to a large nerve distributing station, the sphenopalatine ganglion. Little troubles here give more distressing symptoms than in any of the other paranasal cells. I say little troubles, because it is so difficult to discover any definite pathology, and still when properly treated, its response is often times almost dramatic.

When a patient comes to me complaining of a deep dull distress in the head, which at times sharpens into definite pain, traveling either toward the occiput, the mastoid process, the back of the neck or the corresponding shoulder, I think of the

sphenoid. Again, it may extend to the temple or to the bridge of the nose, spreading over the area covered by the second division of the trigeminus. Now add to this a postnasal discharge, a frequent slight sore throat, which extends high up and well back. These patients will tell you that it is not painful for them to swallow, but that the "soreness" is higher and that it frequently radiates to the ears. This may be accompanied by tinnitus and sensitiveness to loud, sharp sounds. I have noticed that these latter symptoms are more marked toward evening, when the patient is fatigued from a hard day's toil. There are other characteristic symptoms, such as dryness in the nasal chambers alternating with periods of turgescence and spasms of sneezing, following rapidly one after the other, and coming on any time of day or night. I have a patient under my care now whose only complaint was sneezing spells followed by nasal obstruction, which came on quickly and was almost complete for long periods of time. She had a "hot" feeling through the bridge of the nose which extended back deep under the eyes. She responded quickly to treatment of the sphenoid sinus.

With posterior rhinoscopy and Holmes' nasopharyngoscope the posterior sphenoid wall will show a thickened, dark red, inflamed mucous membrane, which extends over the anterior surface and well up toward the superior concha. The pharynx will show red lateral streaks extending well down to the base of the tongue.

These patients will give a history of general nervousness. They sleep poorly. They have difficulty in keeping steadily at one task. Their power of concentration is poor. This is particularly true of those who have had no trouble in remembering before. They have early fatigue muscle and joint pains and restlessness. They are unable to take part in any social function without forcing themselves to do so, and when they do indulge, they are completely exhausted for two or three days following. I believe, without exception, that they all have painful vision. They have all been fitted with glasses but their asthenopic symptoms continue.

The treatment varies with the kind and severity of the infection. If they have not been treated before and if there is no

evidence of an empyema, I usually subject them to medical treatment in the office. This particularly holds true in those cases where the sphenopalatine ganglion is irritated and its nerve distribution giving local symptoms. I attempt to promote better drainage by shrinking the mucous membrane in the postnares by bathing both the anterior and posterior surfaces with some antiseptic, nonirritating solutions, such as the milder silver salts. When I use a tampon in the upper and posterior nares I usually use a 10 per cent solution of neosilvol. In my hands, it has given just as satisfactory results as argyrol. The great advantage it has over argyrol is that it is so much cleaner. When argyrol is used freely on tampons, it keeps the operator's hands, the patient's face, the office trays and linen so stained that I consider it impractical.

A great many of these patients improve rapidly, and after nine to twelve treatments, are well again. There are others who make some improvement but soon relapse. Some of them make no improvement whatsoever, in fact they are made worse. I have often noticed that satisfactory results will be obtained by a course of treatments, only to be followed in a short time by a complete return of former symptoms. This will occur time after time. There are others, who, with the slightest exposure to cold, will have a recurrence. I have often had very gratifying results by instructing these patients how to dress and how to avoid exposure both at home and at their work. I tell them they must not get chilly, for just as soon as the body begins to feel cold and chilly sensations creep over it, just that soon a return of symptoms will be experienced. These unfortunate people will all be benefited by an extended vacation to a warmer climate, but when for obvious reasons this cannot be taken, I do a complete sphenothmoid operation. Most of them will recover after the nasal mucous membrane has again healed over and the function of the nose returned to normal. When the trouble is caused by an irritation about the sphenopalatine ganglion, I cocaineize the ganglion and follow by an application of silver nitrate. This procedure often brings about most pleasing results. Relief is obtained out of all proportion to the treatment applied or the actual pathology observed. The following case will illustrate:

Mrs. C. T., an American, housewife, married, age 36, came to my office January 22, 1922, complaining of an inability to read. Every time she made the attempt, the page would blur and in a few moments would be followed by a violent headache, accompanied by dizziness. She told me that it had been eight years since she had been able to read anything other than the larger headlines in the daily papers. She had been repeatedly fitted with glasses without relief. A careful refraction with cycloplegic showed the vision normal. I made a diagnosis of sphenopalatine irritation and cocaineized the ganglion with a 10 per cent solution followed by an application of 2 per cent silver nitrate. After the first treatment she improved so that she could read large print without discomfort. I gave her three more treatments at four day intervals, after which her symptoms entirely disappeared. She has had no difficulty in reading since. Many similar cases could be mentioned.

For a number of years I have been concerned about the "silent" infection in the maxillary sinus. There is no place that symptoms so remote are so constantly associated with definite pathology and which, when gone into thoroughly, can be connected with the actual distant symptoms of the patient. One would think, where an area so large as that of the maxillary sinus is involved in an inflammatory process, and this so closely associated with large sensitive nerve trunks, that symptoms would be definitely located about its walls or in parts contiguous thereto. This is not borne out by facts. My first question when taking the history is: "Of what do you complain?" I have selected from my records eighty-five cases whose antra I considered serious enough for operation. These cases were not chosen because of the complaint, but were taken one after the other in numerical order over a definite period of time. It includes adults only, since children, as a rule, do not make definite complaint about anything. For three years I have attempted to put on my records, under the heading of "Chief Complaint," just what the patient tells me he is consulting me for—not what I elicit during the history taking, but rather in his own words, why he seeks my advice. In the eighty-five cases I found there were twenty-four complaints. I venture to say this is much greater than in most other diseases, unless the chronic, degenerative diseases, such as gen-

eral syphilis, tuberculosis, etc., are considered. The following table will give you some idea of what I am attempting to show you:

Complaint	No. of Cases
1. Profuse purulent discharge.....	14
2. Frontal headache	12
3. Nasal obstruction	10
4. Tinnitus	5
5. Chronic red eyes	4
6. Tonsillitis	4
7. Frequent colds	4
8. Rheumatism	3
9. Cough	3
10. Vertigo	3
11. Gradual deafness	3
12. Asthma	3
13. Maxillary headaches	2
14. Hoarseness	2
15. Hay fever	2
16. Blurring of vision	2
17. General malaise	2
18. Asthenopia	1
19. Diplopia	1
20. Hemianopsia	1
21. Ulcer of cornea	1
22. Chronic purulent otitis media.....	1
23. Otagia	1
24. General nervousness	1

You will note the greater number of cases, fourteen in all, or about 16%, complained of profuse purulent discharge. In most of these cases this meant anterior discharge, the use of the handkerchief. Some of them said they had to clear the throat very frequently, but they were very few. The discharge blown from the nose is so evident that they usually do not think of the posterior escape until it is called to their attention by the examiner. There were two cases among these who definitely complained of a bad odor and a disturbed taste. This greater percentage is not surprising, for given a chronically infected antrum, we would think of increased discharge from the nose.

The next highest number of cases, twelve in all, or about 15 per cent, was frontal headache. These headaches varied somewhat. Some of them were unilateral, corresponding to the same side as the involved antrum. A number had pain extending over the entire brow and some of them through the

eye and under the orbit. The X-ray showed all these frontal sinuses clear. A few of these headaches could probably be accounted for by the general toxemia, the absorption coming from the infected antrum, but I think a greater percentage was due to a closure of the natural opening in the middle meatus which drains both the frontal and the antrum.

The next number of complaints, ten in all, or about 10 per cent, gave nasal obstruction as their main discomfort. This symptom can well be explained by the swollen nasal mucous membrane caused by the irritating discharge. It may also be caused by polyps and polypoid degeneration of the turbinate body with its erectile tissue covering.

The number now falls quite abruptly to five cases, or 5.5 per cent, these giving as their greatest annoyance tinnitus or ringing in the ears. This symptom is no doubt produced by an involvement of the eustachian tube and can usually be traced back to the beginning of the antrum infection.

Under the next three complaints are four cases each (1) chronic red eyes. I have classified here those individuals whose first discomfort was redness, burning, itching, roughness inside the lids and photophobia. Usually they have pterigia or pterigumlike growths of the conjunctiva extending from the inner canthus toward the limbus. They have used all kinds of washes, they have been treated for "granulated lids" and most of them have had glasses without relief; (2) tonsillitis and frequent sore throat. Under this heading I have grouped those cases who complain of soreness high up in the back of the throat. It comes on at frequent, irregular intervals. Some of them have swollen, bright red tonsils and painful swallowing. Their cervical glands are tender and enlarged. One man noticed this when he took his morning shave. This is the sentinel gland that so frequently enlarges and gets tender to touch with each cycle of lymphatic involvement. Left untreated and carefully observed by the patient himself, he will tell you that these lymphatic attacks with sore throat occur about every ten days to three weeks. (3) Frequent colds was the complaint of four patients. With the least exposure they catch a fresh cold or they have a cold all the time. They are never free from some annoying symptom which makes them feel that they are either just starting with a cold, or that if

this or that exposure is not definitely avoided they will have another.

Following this there are five complaints, three cases in each.

I was rather surprised when I found that only three cases had complained of "rheumatism." I have called this muscle and joint pain because none of them had any changes in the joints which could be found clinically. They complained of a deep aching pain, first in one set of muscles and then in another. They all complained of pain between the shoulders and the back of the neck. The thing that strikes me about these pains is their fleeting character. They cause great suffering for a short time, then suddenly they are gone. The patient says he has "rheumatism," but what he really has is a myalgia or a painful impulse coursing through a sensory nerve and its branches.

Cough, usually productive, worse when lying down and at times almost intractable. One patient had coughed for six years. She had been treated for almost every lung and laryngeal malady. The condition stopped when a double Caldwell-Luc had been done and a purulent nasal discharge cleared up.

Vertigo.—I had thought this condition more common than I found it. I believe it is noted more frequently in the acute cases.

Gradual deafness is not so often complained of. It is frequently found when an examination of the ear is made.

Asthma.—The three cases here come with this complaint only. They were benefited by operation. One has had no further attacks. It is now nine months since he was operated upon. The other two still have an occasional asthmatic spell, especially when they have a cold, but the attacks are not so severe and they are further apart.

We now have five more complaints with two cases each.

Maxillary Headache.—I have grouped here patients whose complaints were in the region of the second division of the fifth nerve, particularly that area directly over the infraorbital foramen and in and about the upper bicuspid and molar teeth. I was surprised to note that there were not more. Here again this symptom is very common in the acute form. It will usually be found somewhere back in the history.

Hoarseness.—One of the patients was a singer, the other a school teacher. Both used their voices more than is usual, and both must use them to carry on their work. Hence they were more annoying, and relief was sought more urgently than in the same condition in the patients with other vocations.

There were two cases whose only complaint was hay fever. If we give these patients a careful examination, we often find the trouble in the nose or the paranasal cells.

Blurring of Vision.—One case could see only shadows, this in the eye corresponding to the infected antrum. Here the ethmoid was probably also involved, but the maxillary infection was so marked that only this sinus was opened. Of course, better drainage was promoted by postoperative treatments of the antrum. He had a retrobulbar neuritis and hemorrhages in the retina.

General Malaise.—There were two cases who complained of nothing but a general ill feeling. They suffered with early fatigue. They had no ambition and found it difficult to accomplish any task.

Finally, there were seen more cases with an equal number of complaints—asthenopia, diplopia, hemiopia and ulcer of the cornea—all definitely in and about the eye and the visual mechanism. The patient with ulcer of the cornea had had the diagnosis made by her family physician; however, this was her only complaint when she came to me.

One case had chronic purulent otitis media. The ear got entirely well when the antrum was treated and the patient instructed to stop blowing the nose.

The series is concluded by one case of otalgia or pain in the ears, while another complained only of general nervousness.

I think it is only fair to the patient to let him first have his definite say in his own way of expressing it. It is always interesting to note how near he comes to what you afterwards find in the course of an examination.

I attempt to make a diagnosis of chronic maxillary sinus infection by:

1. Nasal inspection and transillumination.
2. Needle puncture and nasal washings.
3. The roentgen ray film.

The transillumination is of diagnostic value only when the findings are positive and unilateral, and the shadow under the eye corresponds to the nostril in which inspection has shown pus, but if the shadow is bilateral, I consider it of no value unless corroborated by the second diagnostic procedure, the needle puncture and washings. I puncture through the nasointral wall with an ordinary curved trocar (Charlton's). To this is quickly attached a Fieck one piece syringe, and with the nurse operating the syringe with firm gentle pressure, the antrum is readily and easily washed out. I note the odor and physical characteristics of the pus, whether it is in large, compact, thick quantities or if it is flocculent and well distributed throughout the washings, whether mucoid or mucopurulent. If these washings show but little purulent material and are in small flakes, well distributed, I advise them to return to the office eight to twelve times for the same procedure. Most of these cases will improve under such treatments. They usually have no return of the trouble. On the other hand, if from the diagnostic washings, I find a mass of pus that comes out at one time after considerable pressure has been used on the syringe, I try the same procedure but advise the patient that, if after a half dozen treatments the condition shows no improvement, he should be operated upon. Now, again, if the washings show a more liquid, green, foul smelling discharge which mixes well with the water, I say at once that office treatments will do no good, and that they must subject themselves to more radical surgical measures.

In private practice it is not always possible to have an X-ray film. Some of the patients cannot afford it, but when possible, it should always be made use of. I think it always helps in the borderline cases. It is of decided value in negative sinuses, and it goes without saying that it is of great service in the positive ones. It shows the size, the shape and pictures the abnormal. It should always be used when making a diagnosis of frontal sinusitis, for here we have the greatest number of anatomic irregularities. If the film shows but little blurring of the outer margins and over the floor, I treat these patients by conservative measures, such as cleaning out the normal opening, infracturing the middle turbinates, keeping the middle meatus clean and removing obstructing septal spurs and deviations. They

usually get well. If the film shows the cavity consistently cloudy throughout, I open these antra and attempt to promote better ventilation and drainage. It is seldom that they get well with anything less radical.

These are not hard and fast rules. I am governed somewhat by the general physical condition of the patient, by the age, the sex, the occupation and mode of living. Pregnancy varies the therapeutic procedure. I frequently find myself trying to make the treatment fit the surroundings and environments of the patient. I tend more toward the operative side for those patients who live long distances away and whose circumstances will not allow them to come to the office for a great many treatments. We are a long, long way from giving these patients ideal prolonged care, which they need.

In concluding, let me say that seeking for infection in these little dark chambers is difficult indeed, when there is only one sinus or one group of sinuses involved. Now, if we add to this infection in the alveolar processes, in the tonsils, the middle ear and the mastoid, we cannot be too careful in taking the history and in making the examination. Correct diagnoses are in proportion to the amount of time and careful study we give our cases. Rational therapy cannot be administered if we do not properly interpret the symptoms and findings. When we reach the stage that we can cure chronic sinusitis, we will be benefactors in all that the word implies.

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LV.

CASE REPORTS: ONE OPAQUE AND TWO NON-
OPAQUE FOREIGN BODIES IN THE
RIGHT MAIN BRONCHUS.*

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Case No. 1 is presented, as it is of interest due to complications which developed. The question of management in this case is the chief point of consideration. The histories are as follows:

Patient, child, age three years, had the following history: Was admitted to the hospital December 16, 1926. Baby had "asthma" six weeks ago. Three weeks ago patient became quite sick, and the condition was diagnosed by the family physician as pneumonia and so treated. Child's condition did not improve, and on December 13, 1926, an X-ray of the chest was made. A tack was shown in the right chest. The parents did not know when the tack was aspirated. No history of sudden coughing or strangling was given.

Physical examination on admission: Left lung negative; right lung, intercostal spaces practically obliterated; chest up to mid-intrascapular region flat; apical and bronchial breathing; marked impairment of respiratory sounds. X-ray examination revealed the presence of a mattress tack in the right main bronchus. The temperature on admission was 104° , respiration 50, pulse 160. Blood picture, W. B. C., 26,000; hemoglobin, 50 per cent; polynuclears, 77 per cent; lymphocytes, small, 23 per cent; urinalysis negative.

Consultation with the internist brought up the question as to whether we should first remove the tack bronchoscopically or to drain the pleural cavity by means of a thoracotomy. In view of the critical condition of the patient, we felt that the empyema, which was, no doubt, secondary to the aspiration of

*Read before the American Bronchoscopic Society, Atlantic City, May 21, 1927.

the tack, had become the more pronounced symptom and should receive first consideration. There was difference of opinion as to this procedure, that being my reason for presenting this case to you.

The child died 36 hours after the thoracotomy had been performed. An autopsy was not permitted.

Case No. 2, patient age 4, was admitted to the hospital, January 14, 1927. The following history was given: The child suddenly strangled after eating parched corn four days previously. The child had a number of severe coughing spells immediately after the aspiration of the corn. Has coughed some since, and breathing has been of the "wheezing" nature, except when the child is asleep. X-ray examination of the chest did not reveal the presence of any foreign body or pathology of any consequence in either lung. Physical examination negative both lungs, with the exception of the wheezy breathing.

Temperature on admission 98.9°, pulse 100, respirations 30. Blood count, W. B. C., 11,600; hemoglobin, 65 per cent; polynuclears, 62 per cent; small lymphocytes, 37 per cent. With a history so strongly suggestive of foreign body aspiration, we decided to pass the bronchoscope. There was a small amount of secretion in the trachea and bronchus. This was removed and a portion of parched corn was seen in the right main bronchus. It was not impacted and its removal was easy. The child had no postoperative reaction, and left the hospital on the second day in good condition.

Case No. 3.—Age three, was admitted to the hospital March 14, 1927. The following history was given:

Five days ago the child suddenly strangled while eating a grain of corn. Coughing was profuse at the time, and since then has had a slight wheezing and rattling in his throat. Parents state that the child became very "feverish" the day following the aspiration of corn and has remained so.

Temperature on admission was 103.2, pulse 140, respiration 45. Physical examination of chest showed dullness and diminishing breathing sounds over the base of the right lung. Left lung was negative. X-ray did not reveal the presence of a foreign body, but showed an increased density over the base of the right lung. Bronchoscopy was done March 14, 1927. The mucous membrane of the larynx, trachea and bronchus

was very much inflamed and a considerable amount of mucopurulent material was present. After this was removed, a portion of a partially masticated grain of corn was found in the right main bronchus. The corn was removed in pieces. The laryngo-tracheo-bronchitis, which was present at the time of the bronchoscopy, was aggravated by the instrumentation. This was evidenced by the increased dyspnea, which was to such an extent that tracheotomy was necessitated within twelve hours. The tracheotomy partially relieved the dyspnea. The secretions were profuse and tenacious. So much so that it was necessary to introduce a small catheter into the trachea and bronchus several times daily for the purpose of aspiration. The child remained in the hospital ten days. During this time fine particles of corn were observed in the bronchial secretions. Five days following the operation, the temperature ranged from 100 to 104 degrees, and as the secretions became less the other symptoms subsided proportionally. The tracheotomy tube was removed on the eighth day and the patient was dismissed on the tenth day and has since remained well.

Cases Nos. 2 and 3 are ones of nonopaque organic foreign bodies in the air passages. Both were children of practically the same age, and in the sojourn of the foreign bodies were the same. It is very interesting to note that case No. 2 had very little reaction as the result of the aspiration of the corn, which was parched, and case No. 3 developed a very severe laryngo-tracheo-bronchitis. The grain of corn in this case was not parched. It is possible that the parching of the corn destroyed its toxic element.

LVI.

THE SO-CALLED IONIZATION TREATMENT OF OTITIS MEDIA: ITS PHYSICOCHEMICAL ASPECTS.

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Notwithstanding that quite a large amount of literature has appeared on the subject of treatment of otitis media by electrophoresis or so-called ionization, no satisfactory explanation has been given of the probable physicochemical factors involved in this method. At various times a number of different elements were used in the treatment, and various formulæ have been recommended for the ionizing solution, but most of them have been discarded as unsatisfactory for one reason or another. In this paper, these methods and formulæ are analyzed and account is taken for them being discarded or retained, the probable reactions which occur in the middle ear during such electrophoresis are noted, and modifications of the present methods are suggested.

As living cells, whether they be bacteria, leucocytes or other tissue, are the objects toward which attention will be directed to a great extent, something of their physical and chemical properties needs be considered first.

All life processes take place in colloidal systems. Only those structures are considered living which at all times are colloidal in their composition. Since organisms are merely special instances of colloidal systems, there can exist no biologic problems in which colloid chemistry does not play some part. Protoplasm is neither solid nor liquid when compared with typical solids or liquids; its physical peculiarities are those of a hydrated emulsoid which may show all degrees of fluidity ranging from those which, on the one hand, are characteristic of a normal liquid to those which, on the other hand, are characteristic of a solid. Protoplasm is a dilute colloid, uniting within itself the properties of a liquid and a solid; just so does

living matter show properties which at one time make us think it fluid and at another solid. Protoplasm shows, for example, capillary phenomena, protoplasmic streamings, vacuole formation, throws out pseudopods and its separated portions from droplets.¹ All these are properties of liquids. Upon the other hand, protoplasm shows a plasticity and maintenance of form which is seen only in solids.

Kolmer² notes the following as ideal properties for a bactericidal agent: (1) A direct chemical reaction between the compound as administered or after some transformation in the body, with some protoplasmic constituent of the parasite, resulting in death or crippling of the latter by interference with its vital processes. (2) A physical or physicochemical interaction with the protoplasmic colloids of parasites involving precipitation, coagulation, changes in the electric charge, etc., sufficient for destruction or crippling. (3) The possible production of new compounds in the tissues capable of chemical or physicochemical interaction with protoplasmic constituents of invading parasites rather than the production of these effects by the compound direct. In order to choose a substance which will fulfill all or the majority of these requirements, it is necessary to know something of the properties of the colloids of bacterial protoplasm and of leucocytes and the fixed tissues.

The colloids encountered in the body are mostly protein. These proteins exhibit characteristic colloid reactions, such as the Brownian movement, and to a slight extent show the Tyndall phenomenon, and they all follow Hardy's³ rule. This latter deals with the electric charges on colloidal particles. In most cases the particles of colloids possess an electric charge, either of positive or negative electricity. This has been found to depend upon the hydrogen ion concentration of the medium; for example, the hydrogen ion concentration of the blood is 7.4, which is slightly on the alkaline side of neutrality, and its proteins carry a negative charge. This has been accounted for by the theory of adsorption. If colloidal particles are placed in a solution which contains ions, and if there were any means by which ions of one or another sign could be deposited on the surface, it is clear that these charged ions would give to the surface a charge of the corresponding sign. Such a pro-

cess is known as adsorption. In the adsorption of anions or kathions, say those of Cl^- or Na^+ , by a protein molecule, the resulting product is regarded as the loose combination of such ions with the potentially free valencies of the COOH or NH_2 groups of the protein molecule. If the reaction takes place in an acid solution, in the case of adsorption of sodium chlorid by a protein molecule, the chlorid ion would unite with the NH_2 group of the protein, whereas in an alkalin solution the sodium ion would combine with the carboxyl group. This combination of the sodium ion of sodium chlorid with the carboxyl of the protein is probably what occurs in the case of body proteins, as it has been noted that the reaction of the plasma which comes into contact with all the cells of the body, is slightly alkalin. Loeb⁴ gave quantitative proof that proteins combine with acids and bases in stoichiometric proportions. Furthermore, he has shown⁵ that, owing to the presence of the amino group in the amino acid molecule, this reacts with acids as though it were a basic substance. When placed in an alkalin solution, amino acids behave as though they were acids, because of the carboxyl group. Since the protein molecule likewise has at least one free amino group and one free carboxyl group, it will yield a protein kation in the presence of acids and will form protein chlorids, etc. In the presence of bases it will yield a protein anion to form such compounds as sodium or potassium proteinates.

When the sodium ion bearing a positive charge unites with carboxyl groups of the protein, this charge is neutralized; but it has been found by Helmholtz⁶ that the presence of a charge on a surface implies that of an equal and opposite charge in the immediate neighborhood. Therefore, in order to account for the negative charge which a protein colloid is known to possess in the presence of sodium chlorid in alkalin solution, it is necessary to assume that the negatively charged chlorid ions are held very close to the protein molecule by the electrostatic attraction of the sodium ion. This is the so-called Helmholtz double layer, and gives to the colloid under these conditions its negative charge.

It is well known that when a direct current is passed through a solution containing an ionized salt, the positive ions or kations move toward the negative pole (kathode), whereas

those bearing a negative charge (anions) pass to the positive pole or anode. Here they give up their charges and are deposited, or react with the electrode or with the solvent. When it is recalled that the body proteins bear a negative charge, this will account for the migration of leucocytes to the anode when present in an isotonic solution. That most bacteria are likewise electronegative is probably attributable to the same adsorption phenomenon.

These phenomena of attraction of colloids to the poles of a source of current can be applied to one of the problems encountered in the treatment of suppurative otitis media. As has been noted, leucocytes and the majority of bacteria possess a negative charge and are consequently attracted to the anode, and this electrode should be the one placed in the external auditory meatus during the passage of a current. Any condition producing leucocytosis may increase the parasitocidal activities of the blood, especially if phagocytosis were promoted (Kolmer, l. c., p. 25). This is of especial importance in the treatment of such a chronic infection as that of the middle ear in which the bacteria are almost sure to have taken up a position of great strategic strength in the depths of cells and especially in submucous tissue. Thus, beside attracting bacteria to the surface where they may be reached by the bactericidal drug, it attracts phagocytic leucocytes to the vicinity and prevents the carrying to adjacent tissues of leucocytes bearing bacteria which may infect such tissues. In connection with the consideration of the electric charge on bacteria, the observation is significant that the reaction of pus serum is usually alkaline, becoming strongly alkaline if much ammonia is produced, which occurs if there is secondary infection with organisms of putrefaction. Presumably, the nature of the reacting organisms will modify the reaction. For example, *Bacillus pyocyaneus* and *Streptococcus hemolyticus* cause alkali formation in pus, whereas some staphylococci cause acid formation. The pneumococcus is said to cause pus to become markedly acid⁷. The results of these varying reactions become significant when considering the clinical aspects of the ionization method.

One of the questions which has heretofore been vigorously discussed is that of which metal or other element to use in the ionization treatment of suppurative otitis media. At various

times the following elements have been used for this purpose: Magnesium, iron, aluminum, copper, zinc, iodine. Of these, zinc is the only one which has thoroughly withstood the clinical tests and at present is the only one in extensive use. The reasons for retaining one and discarding the others are obtainable from information regarding their reactions under the conditions to which they are subjected.

It has been known for a number of years that the properties of an element depend to a great extent on its atomic number. Of the metals considered in the preceding paragraph, magnesium has an atomic number of 12, aluminum 13, iron 26, copper 29, and zinc 30. The electrolytic solution tensions of these metals increases with increasing atomic number, becoming more positive from left to right; their ionic speed, adsorbability, toxicity and effectiveness in precipitating negative colloids increases in the above order. Mathews⁸ found that the physiologic properties of an ion depend on its electric state (valence) and electric stability (electrolytic solution tension). It has been noted that this latter property increases with increase of the atomic number. For example, the electrolytic solution tension of copper is -0.3488 volts, of iron 0.045 volts and of zinc 0.1781 volts.⁹ Thus it will be seen that of the metals considered, zinc is the most chemically active.

In the clinical experiments with the various metals it was soon learned that magnesium and iron could find no place in middle ear electrotherapy. Magnesium is decidedly destructive to the tissues, as is shown by its use in the removal of warts and similar growths. Iron stains indelibly the tissues to which it is applied under influence of an electric current. This leaves copper and zinc, with atomic numbers of 29 and 30 respectively, to contend for the position as the most effective metal for use in ionization therapy under the conditions encountered in suppurative otitis media. From the data presented herewith it will be observed that zinc has slightly the advantage. The equivalent conductance of copper ions is 45.9 and of zinc 47.0 ; thus more current is carried by a zinc ion than by one of copper; the electrolytic solution pressure of zinc is less than that of copper, permitting it to form ions more readily than the latter. The relative ionic speeds of copper and zinc are 47.16 and 89.0 respectively, thus the copper ion moves

more slowly per unit of current. These physical differences are relatively insignificant compared with the chemical properties of the two metals.

It will be recalled that in the conditions under which protein colloids are encountered in suppurative otitis media, the colloidal particles have sodium ions adsorbed to them, leaving free the chlorin ions. Inasmuch as this adsorption of sodium chlorid appears to be a phenomenon normal to such colloids, it must be the purpose of a therapeutic ionizing method to so alter the nature of the adsorbed layer or even the chemical composition of the entire colloid, that the cell may no longer function as a viable unit. Of course, the substance of the cytoplasm is in all cases, without any exceptions, a colloidal substance. Consequently, the reciprocal action between the cytoplasm and the medicinal ion will always be a heterogeneous colloidal process. Since the reaction takes place in a heterogeneous medium, it is found to occur in two stages, namely, the physical contact of the reacting ingredient and the cell plasma (adsorption) and the chemical reaction between them.

When the physical contact between a given preparation and the cytoplasm is attained, a chemical reaction between them inevitably follows (Kolmer), just as the formation of salts is inevitable when any acid and base fall into mutual contact. Such is the action of copper and zinc. The ions of these metals are adsorbed to the protoplasmic colloids by replacement of the sodium, from which position they eventually combine chemically with the carboxyl radicals of the protein molecules, forming compounds which are relatively less soluble than the compound with sodium as which they occur in the conditions being considered, namely, a colloidal protein solution in the presence of ionized sodium chlorid in an alkaline medium. The displacement of a charge on the surface of a colloid particle is possible by electrical adsorption only when the valency of the oppositely charged ion is greater than that of the ions to the primary adsorption of which the surface owes its charge.¹⁰ When the oppositely charged iron is acted upon by the chemical affinity of the surface atoms, a reversal is possible independent of the valency of the adsorbed ion. Such is the case in the adsorption of sodium ions by the carboxyl group of the protein. But the

alkali metal kations can never reverse the charge of a colloidal surface as they are univalent.

It is probable that the action on the colloidal particle by copper ions or zinc ions in solutions of cupric sulphate and zinc sulphate, respectively, is by replacement of the sodium ion by an ion of the bivalent metal, the resulting adsorption layer being more stable because of the relatively smaller solubility of the compounds of copper or zinc with the carboxyl group of the proteins. On account of the relative insolubility of these compounds, the action of copper and zinc salts without the aid of a direct electric current must be only superficial, for the particles soon become coated with the nearly insoluble compound and the action ceases. It was noted previously, that without the aid of the current, those bacteria which are embedded deep in the tissues would not be brought into contact with the bactericidal preparation. At any rate, the effect is comparatively slow without the electric current, as far as chemical behavior is concerned. It is likely that the metal ions are attached at first principally by adsorption, for with zinc sulphate Isgarishev and Titov¹¹ found that in a normal solution of that salt containing 0.25 gm. of protein per liter each protein molecule must have attached to it about 12,500 ions of zinc, whereas only 750 ions per molecule could be accounted for by chemical combination. The protein molecules were not visible under the ultramicroscope but became plainly visible on the addition of the metallic salt, indicating that each particle must have increased in magnitude as a result of adsorption of metallic ions. Isgarishev and Mlle. Ponomareva¹² observed that the degree of adsorption of zinc ions was greater than of copper ions.

Another point which tends to eliminate the use of copper for middle ear ionization therapy is the relatively greater solubility of its protein combinations. Soluble copper salts of the amino acids are well known (Reichert), and the reaction of copper salts with most proteins in alkaline solution is well known as the biuret reaction. This latter reaction is due to the formation of an easily soluble copper compound of biuret (Schiff). Kober and Haw¹³ studied the copper complexes and classified some 144 of them according to their coloration as determined by their copper content and arrangement. The

majority of these compounds were found to be soluble. Serum proteins have been carefully examined as to their precipitation by copper salts,¹⁴ it having been found that serum and albumin solutions free of electrolytes are not precipitated by cupric salts and that precipitation occurs in the presence of sodium chlorid only when the concentration of this salt is over 0.1 N. In the presence of a little alkali the precipitate is dissolved on further addition of the cupric salt. The precipitation of milk protein by cupric salts yielded similar results.¹⁵ These observations support the contention made herewith that the primary adsorption of a simple univalent kation by the protein molecule is necessary for union of the bivalent ion with the protein by displacement of the univalent one.

An investigation of the solubility of the organic salts of copper and of zinc will reveal that the solubility of the zinc salts is in general smaller than that of the corresponding copper salts. It has already been noted that most of the protein copper complexes are soluble in even a trace of alkali. This observation must be used as an argument against the use of copper as a metal for ionizing the middle ear, as the copper compounds formed would soon dissolve and leave exposed a layer of tissue which becomes ready ground for reimplantation of infecting organisms. When zinc is used the resulting compounds are relatively insoluble ones, and it is on this account that cases of suppurative otitis media intelligently treated by zinc ionization are not likely to recur. This conclusion is reached on considering the second stage of the reaction, namely, the chemical action of the zinc ion after its adsorption by the protein molecule.

The essential reason for the skepticism of many otologists regarding the practical value of topical application of zinc sulphate in the treatment of otitis media is doubtless due largely to the difficulty of keeping the active constituent in contact with the infected tissues for a sufficient period of time and, indeed, it may not reach some deeper parts at all. Merely to apply the substance on gauze is usually insufficient, as this may only protect against secondary infection without appreciably influencing the existing infection. In the acute cases the organisms are more superficial and, therefore, of easier access than in chronic cases; in the latter the bacteria are

apt to be in the submucous tissues, protected against destruction by this location. It is for these reasons that a substance which does not possess penetrability in any great degree can never prove very successful in the treatment of chronic infections by direct application, as it comes into contact with and destroys only those organisms swept out to the surface by the secretions, many of which are already dead, without being able to penetrate and reach the real offenders deeply and safely lodged in the tissues. It is only by means of the direct electric current that the zinc ions can penetrate into the tissues to a sufficient depth to reach bacteria lurking in the deeper tissues. As the compounds of zinc with the cell proteins are relatively stable and insoluble ones, they decompose slowly and in doing so liberate in an ionized state the zinc which continues to exert its bactericidal action by further combination with cell proteins. It is probable that to this continued action of the metallic ions may be attributed the infrequency of recurrence of otitis media following zinc ionization therapy intelligently applied.

As to the chemical action which takes place in the middle ear during the so-called ionization with a solution of zinc sulphate and a zinc anode, the following explanation is probably a correct one. As has already been shown, the zinc ions in the solution move toward the kathode. They come into contact with colloidal protein-sodium chlorid compounds and probably unite with their carboxyl radicals by replacing the sodium, forming relatively insoluble compounds. The sulphate ion, being negatively charged, moves to the anode, the zinc wire, and there a reaction takes place as follows: As water is slightly ionized, it combines with metallic zinc to form zinc hydroxid; the liberated sulphate ion combines with the hydrogen of the water to form sulphuric acid, which readily attacks the coating of zinc hydroxid on the anode, forming zinc sulphate and water. As hydrogen is liberated, this gas is seen to bubble from the external meatus during an ionization treatment.

The concentration of zinc sulphate in the original solution is of some importance. Thus, Liddell¹⁶ observes that albumin is irreversibly flocculated by 0.05 and 1.0 N zinc sulphate solution; from 1 N to 2 N there is no flocculation; beyond 2 N reversible precipitation begins, reaching a maximum of 4 N. A 0.05 N solution of zinc sulphate contains 7.18 grams per

liter, and a 1.0 N solution contains 143.77 grams per liter (14.37 per cent). An ionizing solution containing 1.5 grams zinc sulphate per liter of water is a convenient one, being approximately 0.1 N.

In our experience it has been observed that otitis media resulting as a sequel to certain bacterial diseases is quite resistant to ionizing methods. This is true particularly of otitis media following scarlet fever. Dick and Dick¹⁷ stated recently that scarlet fever is caused by only two strains of the hemolytic streptococcus. As streptococcus hemolyticus is the predominating organism in suppurative otitis media which yields easily to the zinc ionization treatment, it requires speculation as to why otitis media resulting from any two strains of streptococcus hemolyticus should be more resistant to the treatment than that caused by the remaining strains. An explanation may be found by considering something of the possible arrangements of amino acids in different proteins. With the twenty or more amino acids known to comprise the compounds called proteins, the possible number of distinct and different protein arrangements that might exist is enormous. Abderhalden has calculated that twenty amino acids could form at least 2,432,902,008,176,640,000 different compounds, and this without including the enormously greater number that might be made by varying the proportion of the different amino acids in a single protein.¹⁸ If this variation of the amino acid nature and content of proteins is sufficient to give to them an immunologic specificity, why, then, should not the arrangement of the carboxyl groups of a specific protein, such as that found in the different strains of bacteria, be used to account for the selective action of some metals in combining with them? This is a hypothetical but feasible explanation as to why the strains of streptococcus hemolyticus causing scarlet fever (if such, indeed, be the case), are more resistant to the ionization treatment than are some other strains.

The apparent selective action of a metallic ion for any particular species of bacteria would appear to depend to some extent on the isoelectric point of the specific protein of those bacteria. This point is defined by Michealis¹⁹ as that hydrogen ion concentration at which the dissociation of the ampholyte is at a minimum. Thus, if the hydrogen ion concentration of

the medium in which the reaction between the metallic ion and the protein occurs, is above the isoelectric point of that specific protein, the metallic ion will combine more or less readily, whereas, if the hydrogen ion concentration is below the isoelectric point of the bacterial protein, no reaction will occur. An example of this has been shown by Warwick and Stevenson,²⁰ who found that some twenty treatments by zinc ionization of otitis media showing a pure culture of *Bacillus pyocyaneus* were without effect. It will be recalled that this organism produces considerable ammonia, making the reaction of the medium highly alkaline. By neutralizing the ammonia by means of 5 per cent acetic acid, treatment by zinc ionization was successful. In this circumstance it is probable that the ammonia kept the reaction of the bacterial protein above its isoelectric point, with dissociation at a minimum. As the reaction was changed to below the isoelectric point, the protein dissociation was sufficient to permit combination of its carboxyl groups with the zinc ions.

On recalling that leucocytosis is usually produced and is highly desirable, the question might be raised as to whether the zinc ions would not be destructive to the leucocytes as well as to bacterial cells. The selective affinity of a preparation in respect to one or another kind of cell is explained by the physical properties of the substances—i. e., the degree of dispersion of the given colloid, intensity and nature of the electric charge, speed of diffusion, and so forth. If a substance is given the choice of cells of various microorganisms and tissues, it will react in the first place with those which are able to adsorb it in a given medium faster than the others.²¹

As has already been noted, the chemical reaction can begin only after the preparation is sufficiently in contact with the corresponding cell. Consequently, the coefficient of distribution will depend upon the relative speed of adsorption, or the speed with which the substance penetrates into the cells of both varieties. It follows that the coefficient of distribution is the quotient of V/V^1 , in which V is the speed with which the chemical is adsorbed by the cell plasma of the leucocytes and V^1 is the speed with which the cytoplasm of the given parasite adsorbs the same preparation. If $V = V^1$ —i. e., the speed is alike in both cases—then the preparation will be dis-

tributed equally among the given varieties of cells and consequently both kinds will suffer. When $V > V^1$ the preparation possesses toxic properties, or, as it is called, it is organotropic; and lastly, when $V < V^1$ it can be considered as a parasitotropic preparation. The smaller the quotient of V/V^1 the nearer the given preparation corresponds to the ideal curative remedy. Although no work has been done to demonstrate the parasitotropic action of zinc, it is evident from clinical observation that in the case of most of the infecting organisms in otitis media, the quotient of V/V^1 is less than unity for the adsorption of zinc ions.

Although not presenting a very great breach in the scientific treatment of suppurative otitis media by zinc ionization, the use of glycerin as a component of the ionizing solution is to be dissuaded. Among the various properties of the solvent medium which appears to have a marked influence on the properties of the electrolytic solution, the dielectric constant stands out as the most important factor. As the dielectric constant of the solvent decreases, the conductance of the solution is altered. According to Faraday's law, the chemical action accompanying the passage of a current is proportional to the quantity of electricity passing; thus it will be seen to be advisable that the dielectric constant of the solution be not lowered. It is to be observed, further, that the greater the dielectric constant of the solvent, the greater is the increase in viscosity due to the added electrolyte; and that the ionization of a salt in a mixture of two solvents will have a value intermediate between those of the same electrolyte in the pure solvents, for the ionization of a salt is the function of the dielectric constant of the solvent, and the dielectric constant of a mixture of two solvents is in general intermediate between those of the pure components.²² Consequently, the ionization of an electrolyte under given conditions is greater the greater the dielectric constant of the solvent.^{23 24} As the dielectric constant of water is 81 and that of glycerin is 39.1, the degree of ionization of zinc sulphate in each of these liquids can be calculated. With the dilution with which we are working, namely, 1.5 grams zinc sulphate per liter, the percentage ionization of the zinc sulphate in water has been determined as 26.1.²⁵ From this the percentage ionization of the same salt

in glycerin is found to be 12.6, and in a solvent consisting of equal parts of these liquids the ionization of the zinc sulphate would be 19.3 per cent. Thus the degree of ionization in the glycerin water mixture is only 74 per cent of that in water alone. Then, according to Faraday's law, quoted above, the solution containing the glycerin is only three-fourths as efficient for ionization treatment as a solution of zinc sulphate in water. An additional objection to the use of glycerin is that in the presence of water it is slightly hydrolyzed; in the presence of zinc sulphate the liberated hydroxyl radicals combine with the zinc ions forming the insoluble basic zinc sulphate and zinc hydroxide. This is the white deposit usually found in the bottom of the container in which the glycerin ionizing solution has been allowed to stand for some time.

Another practice in the zinc ionization method of treatment of suppurative otitis media which needs scientific consideration is the type of electric current to be employed. In the majority of clinics, especially in Edinburgh and London, and in many in the United States, the practice is to use dry cells or a storage battery as the source of current, and this procedure is universally recommended by writers on this subject. In 1925, Warwick and Stevenson suggested the use of a direct current generator as the source of induced electric currents for the zinc ionization. The advantages are of the nature of the physical effects of the current rather than any difference in the chemical action. The principal disadvantage of the galvanic current as obtained from dry cells or storage battery is that it readily produces vertigo. The patient complains of feeling dizzy and that it seems as if the world were moving toward the kathode. There is usually nystagmus and inclination of the head to the side upon which the positive pole is placed. There is usually a severe burning sensation when the galvanic current is used. The very term "galvanotherapy," so frequently employed by writers on the subject of the so-called ionization therapy of the middle ear, indicates the prevalent use of this type of current.

The direct faradic current as obtained from a motor generator does not produce the same reactions to a great extent, and it has many advantageous features which make it the choice for use in zinc ionization therapy of otitis media. This direct

faradic current is the only one which may be employed with the expectation of entirely satisfactory results. The current is a rhythmic one, the interruptions resulting from the insertion of nonconducting material between the metallic segments of the armature of the generator. There is but one instant at which the brush entirely covers the segment and another at which it entirely covers the insulated portion. The flow of current from the generator is represented by a sinusoid or regularly undulating curve which has as the crest the point representing the instant that a particular segment is entirely covered by a brush, and at which time there is a maximum of current; the trough represents the point at which the brush is in least contact with the metallic segment and there is a minimum of current flowing, while the declivities and acclivities of the curve depict the brush decreasing its contact with one segment, then gradually increasing its contact with an adjacent one. As the brush is ever in contact with a portion of one or another segment, the current is continuous. In contrast, the curve representing the flow of a galvanic current is a straight line.

The chemical effects of the direct faradic current when employed in zinc ionization therapy have already been discussed. There are certain physical effects which make this type of current the preferable one for use in this method. Vibratory stimulation, like all massage, is irritating. It acts as an irritant to muscles, producing contractions which cause an increased blood supply, slightly increased temperature and increased nutrition. By producing muscular contractions it thoroughly stimulates muscle cell growth and is therefore indicated in the treatment of weak or atrophied muscles. Is not this exactly what is needed in an otitis media of long duration? The sinusoidal current produces a number of rapid contractions, the muscle relaxing as the current reaches the minimum. This produces a gentle massage which is in huge contrast to the rigid contraction produced by a galvanic current obtained from dry cells or a storage battery.

Beside the chemical effects of the various ions in the ionizing solution, the polarity of the current is of importance. It has already been shown that the zinc ions are repelled from the anode, whereas the sulphate ions are attracted. This is

true of all ions, that the positive ones are attracted to the kathode and the negative ones to the anode. This fact has been made the basis of a method employing the negative pole in the external meatus and using a negative ion as the reactive element. By such a method iodine and the salicylates have been introduced into the middle ear—the iodine for its action as a counterirritant and the salicyl ion as a sedative for those patients in whom the ear is sore and painful. From a chemical standpoint this procedure is well enough, but it has been employed evidently without any consideration of the physical effects of the polarity of the current. In the first place, the use of the kathode in the ear in suppurative otitis media increases the alkalinity of the discharge, making the medium a more favorable one for the growth of bacteria, for they are of themselves producing ammonia, which gives to the discharge its alkaline reaction. Suddenly, the negative pole is a vasodilator: it increases discharge and softens tissues and may induce hemorrhage. Compare this with the positive pole in this respect: it is a sedative and a vasoconstrictor, stops hemorrhage and hardens tissue. With this comparison it is easy to see the fallacy of employing the current from the negative pole in the meatus in the ionization treatment with the conditions encountered in the average case of suppurative otitis media.

Needless and absurd as it may seem, a warning is given herewith against the use of an alternating current in ionization therapy of the middle ear. The writer has encountered several otologists who have tried to give ionization treatment by using the alternating current from a 110 volt commercial circuit. As might be expected, there was no benefit to the ear, and the general effect on the patient was very disagreeable. In several such instances this has led the offending otologist to proclaim that the method was without benefit to the patient, besides being very unpleasant and dangerous. To anyone acquainted with the chemical action of the two types of electric currents, it will be readily understood why an alternating current may not be used for ionization therapy of otitis media. In the alternating current, as the name indicates, the direction of the current is first one way and then another, the electrodes thereby changing their polarity. If the first alternation is positive as it goes into the solution it starts the zinc ion in the direction of the kathode—i. e., into the tissues. It scarcely gets

started when the following negative wave neutralizes it, with the result that there is no permanent chemical change produced, but merely a chemical irritation due to the change in the reaction from acid to alkalin, and vice versa. Such a current is a tonic for stimulation, but does not overcome disease. The fallacy of employing such a current in the treatment of the disease under discussion will readily be understood.

Believing that much of the unsatisfactory and disappointing results reported by many otologists on the use of the so-called ionization method of treatment of suppurative otitis media has resulted from an incomplete understanding of the probable reactions occurring within the middle ear during such ionization treatment, the writer has given a review of some of the physical and chemical problems entering into the scientific consideration of the method. The infecting organisms are viewed as masses of colloidal protein, and as such are subject to change in their physical and chemical states, due to physical changes, such as hydrogen ion concentration and electric charge on their mass, and to chemical changes in their composition which result from their combination with various metallic radicals with which they come in contact under the conditions observed, such changes so affecting the organisms that they can no longer function as viable units. Several methods of producing these changes are considered: That employing a solution of zinc sulphate, an anode of zinc wire, and a direct faradic current is decided upon as being most satisfactory. The reason for adopting this procedure as most satisfactory is based upon the properties of the zinc ion and of the bacterial proteins under the conditions encountered in suppurative otitis media. As an uninformed otologist is occasionally found trying to give an ionization treatment with an alternating current, the physical and chemical effects of the various kinds of current are given, and the reasons for the suitability of the direct faradic current for the ionization therapy are discussed.

The writer believes that careful scientific consideration of the physicochemical aspects of the so-called ionization treatment of suppurative otitis media will serve to indicate the need of adoption of standard procedure and standard equipment for

this method, and of reports of extensive clinical application of the method under such standardized conditions.

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LVII.

ANGINA LUDOVICI AND KINDRED AFFECTIONS: AN HISTORICAL AND CLINICAL STUDY.*

By H. S. MUCKLESTON, A. M., M. D.,

LOS ANGELES.

HISTORY.

Acute septic diseases of the throat have from earliest times received their due share of attention from medical teachers and writers. The outstanding symptom is suffocation or threatened suffocation: hence are derived the names applied to this class of disease by Greek and Latin writers, *Cynanche* (pronounced sin-ang-ke) and *Angina* respectively.

The etymology of the former word is interesting. Its roots of origin are two Greek words meaning "dog" and "choking." The first root has a parallel in the modern colloquialism, "dog-quinsy." A picturesque variant in Greek is *Lykanche* (wolf).

It may here be noted that the word *cynanche* was taken over bodily from the Greek by the Latin writers for use in treatises, and passed in later course into Old French as *squinancie*, and thence into English as *squinancy*, a form now obsolete, and finally reaches us as *quinsy*.

The Latin term in everyday use was *Angina*, which is itself of Greek origin. The comedian Plautus puts a fretful "aside" into the mouth of one of the characters in the *Mostellaria*: "I'd like to be turned into an angina, that I might seize that she-devil by the throat."

Hippocrates of Cos, the great master-physician of the fourth century before the Christian era, makes many references to *cynanche*. In "The Prognostics" (section 23) he tells of patients with swelling both within and without the throat, of acute affections of the throat in association with erysipelas, of

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swelling and redness determined in some cases outwardly and in others to the lungs with serious prognosis. In his work "Regimen in Acute Diseases" (appendix 2, section 6) there is a vivid description of the tongue, which ". . . changes from broad and becomes round, its natural color turns to livid, from a soft consistence it grows hard, instead of being flexible it becomes inflexible, so that the patient would soon be suffocated unless speedily relieved."

In "The Epidemics" (book 3) there is a case report marvellously concise, and therein worthy of our emulation. "The woman affected with the cynanche (translator, *quinsy*), who lodged in the house of Aristion: her complaint began in the tongue; speech inarticulate; tongue red and parched. On the first day, felt chilly, and afterwards became heated. On the third day, a rigor, acute fever; a reddish and hard swelling on both sides of the neck and chest, extremities cold and livid; respiration elevated; the drink returned by the nose; she could not swallow; alvine and urinary discharges suppressed. On the fourth, all the symptoms were exacerbated. On the sixth she died of the cynanche."

Also in "The Aphorisms" (section 6, 37), we read that "it is a good symptom when swelling on the outside of the neck seizes a person very ill of cynanche (translator, *quinsy*), for the disease is turned outwardly."

Aretaeus, a distinguished contemporary of Galen, living in the second and third centuries of our era, made a distinction between cynanche and synanche; in the former the parts being swollen and beset by other symptoms, and in the latter the parts being contracted with suffocation impending. There is little room for question that the patients in the latter group were victims of epidemic diphtheria.

Galen described, in his "Methodus Medendi" (book 1), a rare species of cynanche, in which the tongue is so swelled that the mouth cannot contain it.

This age was strongly therapeutic. Galen and Aretaeus, Aëtius in the sixth century, and Paulus of Aegina, one hundred years later, give a multitude of remedies to be used in the treatment of diseases of the throat. "And dog's dung, dried and powdered, and rubbed in with honey, is a most excellent

application, more especially the white kind; also the dung of wild swallows, in like manner Cupping instruments or leeches are to be applied to the chin and neck, and the patients must use the stronger gargles from iris, hyssop, gith, southern-wood, liquorice, dried figs boiled in honied water, or in the juice of rue with milk, or mustard with oxymel." (Paulus Aegineta.) Sinapisms, hot fomentations, purgation, and other means of relief form a long list; venesection by both the sublingual and jugular veins is recommended, but laryngotomy, though counseled by an earlier writer, Archigenes, is by these authorities spoken of only to be condemned.

(The translations of the works of Hippocrates and of Paulus Aegineta, of which the writer has been able to avail himself, are those of a Scotch physician, Francis Adams, and were published in 1844 and 1845, the expense of publication being borne, at least in part, by the Council of the Sydenham Society.)

A layman's brief account of the death, in April, 1585, of Pope Gregory XIII, the promulgator of the Gregorian Calendar, is here quoted without apology as worthy of mention, even if bare of medical coloring. The head of the great Augsburg bankers, the House of Fugger, received "through the Antwerp courier" from their Venice agent, the following item of news: "We would inform you that His Holiness the Pope died in the Lord, on the 10th day of this month at 12 o'clock. May the Almighty be merciful to his soul. Within the space of ten hours he was first in good health, then ill, then dead, suffocated by a catarrh. Farnese shows no elation and all those who were sick abed were made to rise through this shock. . . ."

The classical division of diseases by genus and species held its own into the eighteenth century. William Cullen, prominent among the Scotch clinicians, taught that cynanche was a genus with five species, the *tonsillaris*, the *maligna*, the *trachealis*, the *pharyngea* and the *parotidea*. Its characteristics were "Pyrexia aliquando Typhodes; rubor et dolor faucium; deglutitio et spiratio difficiles, cum angustiae in faucibus sensu," which may be translated thus: fevers, at times with likeness to typhus, reddening and pain in the throat, difficulty of swallowing and breathing, along with a feeling of tightness in the

throat. Dr. James Stratton, president of the New Jersey Medical Society in 1789, delivered a dissertation on this subject, embodying in it the teaching of Cullen. Cynanche tonsillaris included simple tonsillitis and quinsy, and also more severe infections with extension to the trachea and suffocation. Cynanche maligna covered faucial and pharyngeal diphtheria, and the mixed infections of scarlet fever and diphtheria. Cynanche trachealis meant croup and laryngeal diphtheria. Cynanche pharyngea was simply an extension to the pharynx of a tonsillitis. Cynanche parotidea was definitely mumps, even the metastatic involvement of the ovary and the testicle being mentioned. However, the distinctions seem not to have been always clean cut, there being some overlapping, for example, of the tonsillar and tracheal species.

Ten years after the delivery of Dr. Stratton's address, the perils of cynanche were brought forcibly to the attention of this country by the illness and death of General Washington.

"Craik and Dick give a short account of the death of Gen. Washington, which resulted from an attack of Cynanche Trachealis. The disease commenced with a violent ague, accompanied with some pain in the upper and forepart of the throat, a sense of stricture in the same part, a cough and a difficult, rather than a painful deglutition, which were soon succeeded by fever and a quick and laborious respiration. He was bled, some twelve or fourteen ounces of blood being taken. Consulting physicians were called in, and frequent bleedings followed, until he had lost some ninety ounces of blood. Vapors of vinegar and water were inhaled, ten grains of calomel were given, succeeded by repeated doses of emetic tartar. Blisters were applied to the extremities, and a cataplasm of bran and vinegar to the throat. Speaking became painful and finally impossible; and without a struggle he died in twenty-four hours after being attacked.

"J. Reid, a London physician, in reviewing the above account, concludes that the General was bled and doctored to death; as none but the strongest constitution could survive such vigorous treatment."

In 1836 and 1837 there appeared a series of articles in the *Wuerttemberger Korrespondenzblatt fuer Aertze*, the subject

of which was a brawny infection of the cellular tissues of the neck. Various names were given to the disease by the authors, such as "Brandige Zellgewebsverhaertung am Halse," "Cynanche cellularis maligna," "Morbus strangulatorius." The writers were all physicians of Wuerttemberg, and had all been extraordinarily keen observers of their patients' symptoms. The leader of the group and author of the first article was Wilhelm Friedrich von Ludwig of Stuttgart, Leibartz (personal physician) to the King of Wuerttemberg. His name lives in the history of medicine. He was born in 1790, and died in 1865. His leadership was recognized by his contemporary, Camerer of Langenau, in that this writer first called the disease after Ludwig, as has been recently stated by Thomas, Ludwig's own description of the disease now called after his name warrants repetition.

"A sore throat of a rheumatic or erysipelatous nature precedes as a rule the first symptoms, which are: slight rise in temperature, with a number of chills, headache, weakness, some loss of appetite, slightly coated tongue, some difficulty in swallowing, which is slight at the early stage, and in some cases scarcely noticeable; then a hard swelling develops, either on both sides of the throat or more commonly on one side, usually in the cellular tissue that surrounds the submaxillary glands, less frequently in that around the sublingual or parotid. This hard growth extends, with some tissue changes, gradually around the throat, then under the jaw to the chin, and down over the larynx, and often backwards into the cellular tissues of the parotid, also causing a marked swelling on the outside. This growth also extends through the cellular tissues that cover the musculature between the larynx and the oral cavity, and even these muscles themselves are sometimes apparently

NOTE.—A compilation of records of epidemics, in this country, of sore throat and of diseases of the throat in general, from the early part of the seventeenth century, and up to the introduction of laryngoscopy, is found in the Proceedings of the First Annual Meeting of the American Laryngological Association, New York, 1879. The first president was Dr. Louis Elsberg of New York, and this exhaustive narrative is a monument to his labors. From this source the writer has gleaned the address of Dr. James Stratton and the account of the last illness of General Washington.

involved in this hardening process; the tongue, deep red in color, rests on the hardened mass thus formed, which is felt as a hard, indurated ring on the inner circumference of the larynx. The ability to open the mouth is much limited, and every attempt is painful; the tongue is pressed upward and somewhat backward; speech is made difficult, and partly because of the pressure on the larynx, and partly because of the morbid changes in the smaller throat muscles, it is rough and not clear, and gurgling. Swallowing becomes very difficult, with straining of all the throat muscles, due undoubtedly chiefly, if not entirely, to the mechanical pressure of the growth, because an inflammatory swelling of the mucous membrane of the pharynx is present either at the beginning of the disease or later if mercurial drugs are used in treatment. Yet with the progress of the disease a considerable amount of mucus gathers in the throat, which is expectorated with difficulty. The skin over the growth appears normal, not reddened, at least in the earlier stages, although stretched according to the size of the growth,—evidence that its cellular layers are not involved in the hardening process. During the first four to six days of development of this local growth, the constitutional symptoms are not marked, the fever is usually very moderate, the strength and general feeling are a little altered, appetite and ability to sleep are not lost entirely, there is some thirst, secretions and excretions are fairly normal. As the disease progresses, some areas of redness of the skin are noted; inside the mouth frequent exudations of inflammatory lymph appear (if this has not occurred before), the growth under the tongue seems softer, as if the serum under the mucous membrane was effused and partly coagulated. On the outside certain areas become softer, sinking in somewhat, and if pressed with the finger feel as if there was air under the skin, or sometimes they become more prominent and fluctuating, as if pus was going to break through, but this does not occur, and the swelling diminishes—or, sometimes at the beginning, and sometimes later in the course of the local process, some spots in the oral cavity, either at the back or sides of the root of the tongue, or more to the front on the under side of the inferior maxilla, break out and discharge a thin, grayish or reddish brown fluid, with bad odor, which becomes more and more like the ichor of

a necrotic process. With the beginning of this process, which is of the nature of real mortification, the general symptoms become more marked. The fever is higher, with exacerbation usually in the morning, the sleep is broken, with heavy sweats, frightful dreams, somnambulism; there is marked increase of sediment in the urine, sometimes slight delirium. Notwithstanding the fact that the growth may diminish in size, swallowing is still very difficult, there are periods of anxiety with fairly free intervals, a nonmechanical but apparently nervous interference with respiration. The symptoms now develop rapidly, simulating the course of a putrid-typhous process, and in four or five days, the tenth or twelfth from the beginning of the disease, coma and death result with evidence of edema of the lungs. There are variations in the symptoms as noted, especially in the development of the fever, the time of the development of the disease and the severity of the local process, which I will not discuss in detail, as my purpose is to give a general picture of the disease that will aid in its recognition at the bedside." (Translation taken from an address of C. G. Coakley.)

In the fifty or sixty years just elapsed there have been many contributions to the subject. Von Thaden, in 1872, published reports of eighteen cases; he regarded Ludwig's angina as an acute bubo of the lymph glands below the angle of the jaw, and advised that the name be given up. Twenty cases were added in 1883 by Roser; he defined the disease as an acute infection of the submaxillary salivary gland and of its surrounding connective tissue, and regarded it as of an epidemic character, with a specific causative agent. The cervical fasciæ received much detailed attention from Koenig (1882), and from Poulsen of Copenhagen (1886). The latter added a wealth of statistics from an analysis of the reports of 251 cases of submaxillary phlegmon, in 22 of which the floor of the mouth was invaded. These writers are quoted at greater or less length by T. Turner Thomas and Ludwig Frankenthal.

The various forms of acute septic inflammations of the throat were reviewed in 1895 by Felix Semon of London. His conclusion was that acute edema of the larynx, edematous laryngitis, erysipelas of the pharynx and larynx, phlegmon of the pharynx and larynx, and angina Ludovici were of a common

pathology, but that angina Ludovici was an affection with local distinguishing marks, but not essentially a separate disease.

Agreeing with Semon, and yet preserving the name as having a very particular connotation, Thomas published in 1908 the most comprehensive study of the subject that the present writer has had an opportunity of studying. His great contribution to our knowledge of the matter is his depiction of the anatomic relationships of the floor of the mouth and the upper cervical regions. His compiled statistics have been used as authoritative by practically all later writers.

Surgical intervention, early and radical, is the burden of almost all the articles of late years. This review of the history of angina Ludovici may well end with a reference to the work of Marschik and others of the present Vienna school, in combating descending abscesses of the neck. Otto Glogau has sponsored for this country the procedure he there saw in operation, and it is to be hoped that his words will bear much fruit. The prophylactic mediastinotomy of Marschik and such radical searching for abscess pockets and brawny induration, at whatever depth in the cervical tissues, as Glogau propounds and carries out, will give life to many for whom grave anxiety is felt.

ANATOMY.

The anatomic relationships which concern us in the study of angina Ludovici are those of the floor of the mouth and the submaxillary space, of the lateral wall of the pharynx and of the fascial planes of the neck.

Two flat muscles are attached to the upper surface of the hyoid bone, the mylohyoid in front and the middle constrictor of the pharynx behind; these two muscles may be regarded as occupying a common plane.

The mylohyoid, triangular in shape, arises from the mylohyoid ridge of the lower jaw, which runs in the inner surface of the bone from the midline as far back as the last molar tooth. Its fibers run in a slanting direction downwards and inwards, the anterior portion to meet with those of the muscle of the opposite side in a strong median raphe; and the posterior to be inserted into the front of the body of the hyoid bone.

This muscle and its fellow of the other side form a muscular diaphragm, the floor of the mouth.

The middle constrictor of the pharynx, fan-shaped, arises from the two cornua of the hyoid bone and from the stylohyoid ligament. Its fibers radiate widely in a backward direction until they merge with those from the opposite side in the median raphe of the pharynx.

Between these two muscles, the mylohyoid and the middle constrictor, is a large gap, the importance of which, as a pathway for infection, has been very strongly emphasized by Thomas. It is occupied by the hyoglossus muscle, which passes upwards from the hyoid bone to enter the side of the tongue by the bend of the submaxillary gland, and by various nerves and blood vessels.

The nerves are the glossopharyngeal and hypoglossal, and the vessels are the lingual artery and lingual vein.

The submaxillary salivary gland has a varying relation to the mylohyoid muscle. From its bed in the groove on the inner surface of the maxilla below the mylohyoid ridge the gland passes backwards to wind around the hinder margin of the muscle, and thence forwards and upwards to project into the floor of the mouth where it lies on the upper surface of the muscle. Thus it has a threefold relationship to the mylohyoid, first below it, then behind it, and finally above it. Wharton's duct emerges from the substance of the gland and runs forward for a distance of about two inches to open into the mouth near the fraenum linguæ.

Where the gland makes its bend to turn around the posterior margin of the mylohyoid muscle it comes into relation with the anterior margin of the middle constrictor of the pharynx.

Both the submaxillary and the parotid glands are covered by the superficial layer of the deep cervical fascia. They approach one another more or less closely, being, typically, separated by the stylomandibular ligament.

In its position on the floor of the mouth, the submaxillary gland is in very close relation with the sublingual gland. The latter lies upon the mylohyoid muscle, as does the submaxillary gland in its intrabuccal portion. Both are covered by the

mucous membrane of the mouth, and both share in secretion of saliva through Wharton's duct.

The extension of an infection from the tissues around the submaxillary gland may, by contiguity, take place along fascial layers or, as Davis states, along the lingual and facial arteries to the sheath of the great vessels.

In terms of fascial routes, the extension may be along the buccopharyngeal fascia covering the middle and inferior constrictors of the pharynx; this is a continuation of the prevertebral fascia, and a pathway to the large connective tissue spaces in which lie the great vessels of the neck.

In cases of infection, traumatic or otherwise, of the walls of the pharynx or esophagus, a cellulitis around these structures may spread forward alongside the great vessels, the connective tissue outside the pharynx and esophagus being loose and immediately adjacent to the vessels' sheath and also to the capsule of the thyroid gland.

Once the great "spatium collimedium" (Cornig) is invaded, there is a ready way of descent to the lower levels of the neck and to the mediastinum, whether the infection be guided by the esophagus, by the trachea or by the great vessels.

(The writer has drawn largely, in the above, from the anatomic study of T. Turner Thomas, the presentation of the cervical fasciæ in Davis' "Applied Anatomy," and Glogau's several articles on descending abscesses of the neck.)

ETIOLOGY.

Statistics as to age and sex are given by Poulsen and by Thomas, and a recent series of 23 cases in the records of the Cook County Hospital is furnished by Yerger. Thomas collected 106 cases; the patients ranged as to age from the earliest infancy to the seventh decade, with the largest number found between 20 and 30 years. Yerger reports patients from 7 to 56 years, the majority in the third and fourth decades. In Thomas' series there were 10 whose sex was not given; of the remaining 96, 76 were males, and 20 were females.

Previous health has no constant bearing on the development of the disease. Eighty-five per cent of Thomas' cases had pre-

viously enjoyed ordinarily good health; of the remainder individual patients had suffered from general ailments, such as privation, gout, typhus or typhoid fever, alcoholism, diabetes or secondary syphilis.

Trauma of the interior of the mouth was judged to be a direct cause in several reported cases. Particular injuries were gunshot wound of the cheek and mouth, wound of the mucosa behind the incisor teeth resulting from a horse kick, an operation for tongue tie, injection of cocain, extraction of carious teeth.

Local mouth infections come in for a large share of blame. Dental caries, involving the molar and premolar teeth, is the commonest of all pathologic conditions mentioned. In 251 cases of submaxillary abscess recorded by Poulsen, 142 had carious teeth. One-third of the patients in Thomas' series had dental caries. Tonsillitis and peritonsillitis come next in frequency, Thomas' figures showing a little less than 10 per cent. The trauma of dental extraction is emphasized as an etiologic factor by Yerger, and this writer adds to other infections that of Vincent's angina. Facial erysipelas, otitis media and externa, ulcers of the lip and nose, and conjunctivitis add a few cases each. The writer will report two cases, one of angina Ludovici, in which no manifest local focus was found, and one of cervical abscess traceable to the swallowing of particles of broken glass.

In one of the earliest reported cases, that of Berman, in 1838, which is the third in Thomas' autopsy series, the initial pathology was caries of a second molar. A hard swelling appeared in the parotid region. A sublingual swelling ensued, with ultimately a descent to the sternum and thorax.

The bacteriology of the disease does not follow the line of a single infection. Streptococci are invariably found, but with them are associated staphylococci, bacilli coli, and in some cases gas producing organisms. The writer obtained serum from a small stab wound of the mucosa of the floor of the mouth in one patient; a smear showed a streptococci only, but a culture gave streptococci, staphylococci and a diplo-streptococcus form, the mixed culture being most likely due to mouth contamination.

PATHOLOGY.

Beginning as a lymph-borne infection from whatever source, the disease implants itself on the cluster of lymph glands lying about the submaxillary salivary gland. Frankenthal views these ten or twelve glands as receiving the lymph flow from the lower part of the face, the gums of the lower jaw, and the entire floor of the mouth.

A streptococcic lymphadenitis and perilymphadenitis leads quickly to an invasion of the cellular tissue about the submaxillary salivary gland, and at this stage Ludwig's clinical course begins. As Ludwig himself noted in his original article, the onset is not always in the submaxillary region. Finger's patient, whose case is No. 5 in Thomas' autopsy series, was in the twelfth day of typhus fever when the sublingual gland and the surrounding cellular tissue were found much swollen. Berman's mention of a parotid swelling has already been referred to, and finds a recent parallel in an article by W. F. Molt.

The anatomic relations of the submaxillary gland lead to a spreading cellulitis. The floor of the mouth becomes involved, owing to the gland being about one-third intrabuccal in position, and the tissues of the neck take part in the extension, being attacked from the superficial two-thirds which lie close under the mandible.

Within the mouth and pharynx the cellulitis spreads to the valliculæ, the pillars of the fauces and the lateral walls of the pharynx. The hard infiltration of the sublingual tissues displaces the tongue upwards and backwards, as noted centuries ago in the writings of Hippocrates quoted above. There may or may not be a true glossitis. There is an actual and very considerable increase in the cubic contents of the mouth as a result of the boardlike infiltration characteristic of a true Ludwig's angina. This increase cannot be compensated for by a relaxation of the floor of the mouth, for the combined mylohyoid muscles form a diaphragm whose descent is physiologically limited, and whose action would be the more constrained should its fibers be irritated by a myositis. The tongue adapts itself to the paths of least resistance; its upward displacement is arrested by the hard palate, so that it is forced to project itself forward between the teeth and backward into the vital

airway of the pharynx, carrying with it the epiglottis and the entire introitus of the larynx.

From this initial stage the process of the infection may be toward healing, by spontaneous resolution, as reported by Thomas in 8 of his 106 patients tabulated.

In the overwhelming majority, however, progress is in the other direction. Boardlike induration, with or without pus formation, is the prominent feature. Frankenthal exposed the suprahyoid region by a median incision, penetrating gradually to the floor of the mouth; then the tissues alongside the sheaths of the great vessels on both sides; then the submaxillary glands, splitting the capsules of both; then the muscular tissues of the tip of the tongue, which he attacked by forcing open the mouth. Wherever he entered, he found no abscess, nothing but discolored serous fluid. His patient recovered, after a tedious convalescence. Lesions of this type are well termed "phlegmon ligneuse," or "Holzphegmon," and call for deep incisions as plainly as does the recognition of fluctuation.

Abscess formation takes place in the majority of cases; Thomas reports that pus was found in 66 of his 106 collected cases. It is to be looked for in the tissues surrounding the submaxillary gland, or about the parotid or sublingual gland in cases where these areas are the sites of the main reaction.

A perusal of many case reports in the literature discovers a tendency for the infection to show itself on both sides of the throat, not simultaneously, not always symmetrically, and not in equal degree of severity. Take, for example, Doig's report of fifty years ago (No. 6 in Thomas' autopsy series): "Parotid, submaxillary and sublingual glands of the left side, much swollen and very painful. On the right side the submaxillary gland is equally indurated, but is not of the same size as on the left side." A very clear illustration is in the case of the medical student in Vienna, which Glogau reported in 1922: "Eight days ago . . . difficulty in swallowing . . . edema of the right side of the neck . . . edema of the right aryepiglottic fold and of the right arytenoid cartilage. After two days the swelling of the right side of the neck subsided and a swelling of the left side appeared that increased continually in size." At the first exam-

ination of a patient whose case the writer is about to report, he found, in association with a swelling of the left side of the floor of the mouth, an edema of the whole anterior surface of the epiglottis and of the right vallecula.

Manifestly the infection can burrow its way through from side to side in the tissues between the hyoid and the larynx. Glogau goes on to report that at operation an abscess was opened on the left side which "reaches into the region behind the M. mylohyoideus, beneath the hyoid bone, corresponding to the pyriform sinus, and that a second portion of it reaches anteriorly to the larynx and into the region of the vallecula." Thomas explains that the extension of a submaxillary cellulitis of one side is probably to the floor of the mouth, along the sublingual sulci and out through the opening in the floor of the mouth on the opposite side, but that more frequently the pathway is along the external connective tissue under the symphysis menti. Three routes of spread are therefore offered.

Below the chin the fascial planes and the connective tissue spaces adjacent to the lingual and facial arteries, to which reference has been made in a preceding section, convey the inflammatory process to the sheath of the great vessels. Lesions of the hypopharynx and the upper esophagus lead to a periesophagitis. If the direction of extension is then forward, an induration or abscess alongside the thyroid gland and the trachea will result. Should the continuation be downward, as will sooner or later be the case, the way to the mediastinum is open, either under the pretracheal fascia to the arch of the aorta and the pericardium or in front of the prevertebral fascia to the pleura and posterior part of the mediastinum.

An illustrative case is given by Poulsen (No. 13 in Thomas' series): ". . . the greatest infiltration is found in the intermuscular connective tissue on the left side of the trachea and larynx close to the internal jugular vein. . . . The process extended downward around the esophagus, where almost to the heart, was found a thick, rather firm, dusky infiltration of the connective tissue, between the mucosa and the muscularis upon the posterior and left side. . ." A pulmonary lesion, assumed now to be a massive collapse of the lung, was detected by radiographic shadow in a case of the writer's. The

shadow was triangular, with the base resting on the left half of the diaphragm. The outline later disappeared.

Pulmonary complications are common in the more severe and protracted cases. Infarctions were found by Gibson (quoted by Thomas), and hemorrhagic and plastic pleurisy by several. "Cynanche is apt to be determined to the lungs" is as true now as when Hippocrates penned his aphorism. The viscid, bacteria-laden saliva and the stridulous breathing invite the onset of an inhalation-borne bronchitis and pneumonia; in fact, the surprising thing is the frequency with which writers report that the lungs remain clear.

A lateral abscess of the pharynx in a case of the writer's drained for three days over the entrance to the larynx, dripping foul pus directly into the air passage. A fetid tracheotomy wound expedited the subsequent bronchopneumonia.

The lymph glandular system plays a relatively inconspicuous rôle. Davis speaks of "a widespread, boardlike inflammation in which all evidence of lymphatic nodes is obscured and there is no outline of any nodes. . . . The disease propagates itself by direct continuity of cellular tissue." Coplin reported having made in two autopsies a complete evisceration of the cervical region, in which the lymph nodes were examined microscopically, and showed practically no infiltration. The lymph vessels, in conveying the primary infection, might be compared to the fuse leading to the high explosive shell, the general cellulitis to which the lymph-borne bacteria give rise.

Between the beliefs in participation and nonparticipation of the lymph vessels, Price follows the *via media*, for his microscopic study of autopsy sections "seems to show that the cellular infiltration has traveled by the lymphatic spaces and by contiguity."

The same writer found in sections that a true myositis was present in the muscles at the base of the tongue. It would be difficult to conceive that the muscular tissue would escape leucocytic infiltration.

SYMPTOMATOLOGY.

At the onset of the disease the patient may or may not give a history of infection in the mouth, such as carious tooth or a tonsillitis, or in the neighborhood. His complaint is of pain

in the floor of the mouth, stiffness in movements of the tongue, pain in efforts to clear the throat, and salivation. Fever is not always present. A point of tenderness is to be expected in the tissues below the jaw, typically midway between the symphysis menti and the angle of the jaw. Swelling appears early in the submaxillary tissues, becoming of a boardlike hardness, and not pitting on pressure.

An increase of the swelling in the floor of the mouth results in a displacement of the tongue upwards and backwards, sometimes later in a forward direction, protruding the organ between the teeth. To swallow or to cough causes severe pain. Breathing is interfered with by the displacement of the tongue and narrowing of the pharyngeal space; the degree of dyspnea varies in different patients and from day to day in the same patient.

The constitutional symptoms are those of a severe toxemia; the fever is intermittent.

The localization of the infection in the formation of an abscess is to be welcomed. Evacuation of pus is often immediately followed by relief of urgent dyspnea, as is shown in a case report about to be given.

The drooling of saliva, consequent upon the inflammation in and about the salivary glands, and upon the extreme pain attendant upon efforts at its expulsion, is inevitable and most distressing.

There are periods of delirium in the more toxic patients. A patient of the writer's had days of delirium, but towards the end his mind was perfectly clear.

A distressing feature of the illness is the utter inability to rest; the patient's wish to rid himself of the ropy saliva, the pain induced by every movement of the head and neck, the ineffectual attempts to clear the throat, all combine to make rest impossible, except if compelled by the administration of opiates.

In a majority of the cases there is a gradual overwhelming of the patient's resistance. Death ends the struggle, sometimes through the respiratory obstruction, sometimes through toxemia, either from the local infection or from the added burden of a streptococcic pneumonia, and in some cases undoubtedly from a mediastinitis.

TREATMENT.

Prophylaxis is obviously to be strongly emphasized. Recalling the high proportion of cases where carious molar teeth provided the initial infection, we ought to bear in mind the potential menace in such teeth and be guarded in approving their retention in a patient's mouth.

Once the disease has reached its florid stage, treatment is supporting and surgical.

The voluntary intake of water is greatly reduced by the agonizing pain elicited by any attempt at swallowing. Retention enemata with glucose, hypodermoclyses of normal saline solution or the intravenous administration of salt solution are to be resorted to. Sedatives are called for, enough to give at least some slight degree of rest.

In recent years there have been vigorous upholders of the benefit of intravenous therapy. D. H. Levy believes that he saved a patient from surgical interference by the use of acriflavin. S. G. Dabney saw a rapid improvement after the use of antistreptococcic serum. The writer's patient received both this serum and mercurochrome 220, but without apparent benefit; however, it is only fair to state that the latter agent was used very late in the course of the disease. Frankenthal is frankly skeptical of the advantage to be gained from the use of chemotherapeutic drugs. Semon ventured the prophecy that in vaccines would be found the most effective remedy; it must with regret be said that his hope has not yet been fulfilled.

The heart is to be sustained by camphor, epinephrin, caffeine and digitalis.

Oxygen should be in readiness.

Hot fomentations are to be used from the beginning, in the hope that, in milder cases, they will favor resolution. And in the more severe they are recommended as a supplement to surgical drainage. W. Harvey has recently advised to "use hot fomentations, changed every fifteen minutes, just as soon as the patient returns from the operating room, and keep on using them until the tissues in the neck start to resume their normal condition."

Except in the rare mild case, early and radical surgical measures offer the only substantial hope of a cure. Thomas, Price, Yerger and Frankenthal describe the operations found effect-

ive. The deep fascia of the neck is to be penetrated, in order to open an abscess or to relieve tension.

Incisions are to be made below and parallel to the body of the mandible through the deep fascia to the depth of the submaxillary gland. Once the deep fascia is reached, the use of the knife is dangerous, and the deeper exploration is to be carried out with blunt forceps after Hilton's method. Glogau's article, quoted above, gives some idea of the depth to which the exploration and dissection is sometimes imperative. The writer has seen a paroxysm of dyspnea, highly perturbing to both the patient and the observer, instantly relieved by the opening of an abscess in this region. To explain the immediate change, one must assume a reduction in the cubic contents of the mouth and a relief of the pressure of the back of the tongue upon the laryngeal introitus.

The swelling below the tongue in or near the median plane and the tautness of the mylohyoid diaphragm call for a median incision above the hyoid bone. Some operators have passed the median raphe of the mylohyoids and split the genio-hyoglossus muscles apart.

The indication for a free incision inside the mouth is not always so clear. However, Coakley states that in an adult patient at Bellevue Hospital there was such evident fluctuation in the floor of the mouth between the tongue and the ramus of the jaw that an incision was made in this region for the evacuation of the pus, and that this incision was all sufficient. He discountenances "the indiscriminate puncturing of an edematous membrane in the aryepiglottic fold, pyriform fossa or side wall of the pharynx," and reports two fatal hemorrhages which occurred within one hour after such "stabbing."

Fluctuation of the side wall of the pharynx is evidence of pus. Where should such an abscess be opened? The writer opened one through the mouth, and felt that he thereby lightened his patient's burden; but the abscess continued to drain into the larynx with a steady drip, and within twelve hours there was so great an increase in dyspnea that a tracheotomy was performed. The writer's present belief is that this abscess lay outside the buccopharyngeal fascia, and had already, when recognized from within, begun to burrow down between the

great vessels in their sheath and the larynx. The better mode of approach would in such a case be by an external route.

This leads us to a consideration of the valuable work of Glogau, who has put into effect in this country the procedure of the present day Vienna school in combating descending abscesses of the neck. In his "prophylactic mediastinotomy," Marschik blocks the approach of infection to the mediastinum by the insertion of iodoform gauze tampons against the sheath of the great vessels at the level of the lower border of the thyroid gland, then by lifting up the gland is able to expose the esophagus and the vertebral column, and by tamponade to interpose a barrier to protect the posterior pathway to the mediastinum. The next step is the drainage of the peripharyngeal or periesophageal abscess, by blunt dissection if possible, but by the use of the knife, if necessary, even in close proximity to the carotid artery.

Such an intervention is bound to avert the indication for a tracheotomy in many cases. But, granted that urgent dyspnea is before the surgeon, the temptation is to defer a tracheotomy until the patient's reserve is all but exhausted. The tube should be ever in readiness, and be introduced in the presence of a persistent and steadily increasing dyspnea.

For these surgical interferences, differing degrees of anesthesia are suitable. Where at all possible, infiltration is to be used, for example in the opening of the deep fascia, or the exploration for abscesses in the submaxillary or midcervical region. Many alarming happenings are reported in connection with the use of a general anesthetic in the dyspneic state. Hervey impresses his readers with his experience; his patient stopped breathing while under light ether narcosis, and the pulse became unrecognizable, but by artificial respiration and the injection of twenty minims of suprarenalin breathing was finally restored and the pulse again could be felt. Glogau, however, in his more extended operative dissection uses light ether anesthesia.

PROGNOSIS.

Thomas' compilation remains the fullest in statistical summary. In 106 cases reported in the literature and met with in his own practice, there was a fatal ending in 43, a little over

40 per cent. Yerger collected, from the records of the Cook County Hospital, 23 cases, a mortality of 43 per cent.

Coakley is more sanguine. "Several cases of this suppurative type are seen each year in our service at Bellevue Hospital, and all have recovered after free external incision into the abscess."

CASE REPORTS.

The writer presents two cases from his practice, one of Ludwig's angina, ending fatally, the other of a cervical abscess, traumatic in origin, ending in recovery.

F. F., an Englishman, aged 50, an actor, consulted Dr. P. B. Roen, on May 16, 1924, complaining of pain under the tongue. The pain had been present for two days, was worse on the left side, and radiated into the neck and up to the left ear. Speech was difficult. There was a spasmodic cough, which hurt the tongue. The patient was not feverish. The sublingual tissues were greatly thickened and tender, and there was a slight injection of the oropharynx. Salt lavage and hot compresses were prescribed.

On May 19th, the writer saw the patient for Dr. Roen. The floor of the mouth was puffy, not tense, and there was tenderness on the left side on counterpressure, most marked internal to the left first molar, although dental caries was not recognized. The tongue seemed large or swollen. The interior of the larynx was free of swelling. The right vallecula was edematous, the left less so, the whole anterior of the epiglottis edematous. Externally there was swelling of the tissues of the neck down to the level of the hyoid bone.

The patient was admitted to the Hollywood Hospital. His temperature was 99.6°, pulse 60 and respirations 18. A snipping of the floor of the mouth for serum allowed a smear to be made; streptococci were found. Swallowing was very difficult, but breathing easy. White blood cells numbered 13,200, with polymorphonuclears 86 per cent.

On May 20th, swelling of both parotid glands was noticed, but there was an improvement in swallowing and speech.

On May 21st, the uvula and left posterior pillar were edematous and were incised, with some relief and some reduction in size, but swallowing was now very painful and difficult.

Antistreptococcic serum was given intravenously (50 cc.) by Dr. Roen.

May 22nd brought a decided increase in the external swelling, which now reached across the midline, and in the tenderness, which was most marked midway between the midline and the angle of the jaw. A second dose of antistreptococcic serum (85 cc.) was given by Dr. Roen. The pharyngeal swelling increased. By evening there was dyspnea. Dr. Alden saw the patient in consultation, and with local anesthesia incised first in the midline above the hyoid bone and secondly below the jaw in the submaxillary triangle; he opened an abscess from which half an ounce of offensive pus escaped. There was an immediate relief of the dyspnea.

By May 24th, the swelling of the left side had gone down appreciably, but there was now an increasing swelling on the right side of the neck, and also a swelling of the right pharyngeal wall. Dr. Alden incised the right side of the neck, at the level of the thyroid cartilage, but encountered only brawny tissue in which was inflamed lymph gland. Later the writer opened a large lateral abscess of the pharynx, which yielded much pus, very foul in odor. He felt at the time that, owing to the external brawny swelling (in a patient with a very thick neck) the internal route was indicated.

Early on May 25th, the dyspnea having increased during the night, the writer performed a tracheotomy. The operation was difficult, owing to the way in which the patient thrashed about in bed, to the depth at which the trachea lay, and to the calcification of the rings. Mercurochrome 220 (25 cc. of a 1 per cent solution) was given in the vein.

On May 26th, Dr. Roen detected signs of bronchopneumonia. His previous repeated examinations prior to this date had shown no change in breath sounds from the normal. The pharyngeal abscess had been draining persistently for two days, emptying its pus just above the level of the arytenoid cartilages. The base of the tracheotomy wound was blackish. The site of the pharyngeal abscess could now be seen more clearly, and was at the junction of the lateral and posterior walls.

On May 27th the lungs gradually filled with fluid. The patient died shortly before noon.

F. P., a boy of six years, was admitted to the Children's Hospital on August 28, 1921. Six hours before admission he had eaten jam and biscuit. The jar containing the jam had been broken and its contents then emptied into another jar. The boy choked in eating, and immediately complained of pain in the throat. A small piece of glass was found in his mouth. Later he complained of pain, both in the throat and in the abdomen.

On admission he held his neck rigidly, and, in fact, all movements seemed to be painful. His temperature was 101.6° , pulse 120 and respirations 38. White cell count was 20,160, with polymorphonuclears 78 per cent. The throat was reddened, and there was swelling of the right side of the neck.

On the following day there was definite tenderness and swelling of the neck, but the swelling was not regarded as glandular. There was dullness over the left lung at the base.

On August 30th, the temperature reached 103.8° with the pulse 140 and the respirations 44. There was an increasing difficulty in swallowing.

On September 1st, he was able to swallow with more ease, but by evening the breathing became labored. Palpation revealed to the writer a hard swelling of the epiglottis and the arytenoid cartilages, and he foresaw that any increase in dyspnea would call for a tracheotomy. The interne administered 5,000 units of diphtheria antitoxin late that evening.

On September 2nd, the dose of antitoxin was repeated soon after midnight. The boy was very restless and showed difficulty in breathing. At 7 o'clock the writer was called, as the boy was in serious straits. Preparation was made for a tracheotomy. No landmarks could be made out, on account of the general swelling of the cervical tissues. Novocain was injected subcutaneously, and a search begun for the trachea. Respiration stopped completely. The writer quickly plunged the knife in the midline, hoping to open the trachea. There was an immediate escape of horribly foul pus. Meantime artificial respiration was being performed. A few minutes after the opening of the abscess breathing was well established. It continued to be labored for about two hours, and then underwent a rapid improvement. Plainly the abscess had displaced the trachea

far to the left, and had worked its way forward between the right lobe of the thyroid and the trachea.

On September 6th, an X-ray of the chest showed a large mediastinal shadow, triangular in shape, with the base resting upon the inner half of the diaphragm.

This was regarded by the radiologist as the outline of an abscess cavity. But the reading of recent papers on massive collapse of the lung has led the writer to believe that the present case is an instance of this condition rather than of abscess. In particular the reader is referred to the work of Churchill and Holmes, who report a series of cases of lobar atelectasis from the wards of the Massachusetts General Hospital.

With drainage, and the use of Dakin's solution, the cervical abscess gradually closed. There was a steady improvement in both the local and the general condition, without any chest findings to warrant anxiety. The boy was discharged on September 17th. The only functional consequence of the illness was a paralysis of the right vocal cord.

By November the outline in the chest had disappeared, as shown in later X-ray plates. The loss of function of the right vocal cord remained. (Recent efforts to find the boy for re-examination have been unsuccessful.)

The writer wishes to acknowledge the courtesy of Dr. Henry Dietrich in assenting to the publishing of this report.

These two cases are reported with a consciousness of short-coming on the part of the writer. He hopes that the lessons they taught him will be of benefit to some of his readers.

In the presence of a widespread cervical cellulitis, the recognition of an abscess in the lateral or lateral posterior wall of the pharynx should lead the surgeon to give serious thought to the mode of drainage. The loose connective tissue spaces external to the pharynx are in close connection with the sheath of the great vessels. A pharyngeal bulging along with a brawny infiltration of the side of the neck is a direct indication for intervention by the external route. D. E. S. Wishart has lately shown the local relationships in diagram very clearly. Glogau has pointed the way to the surgeon in his reports of cases from Professor Hajek's clinic. The procedure followed is to incise along the front border of the sternomastoid muscle

and to carry the dissection by blunt, or, if necessary, by sharp instruments well into the depth of the neck, even to the mucous membrane of the pharynx.

Tracheotomy, to be of greatest service, should not be delayed until the patient's condition is absolutely desperate.

From the second case also the lesson is that watchful waiting may cease to be a virtue. This little patient should have been operated upon some twelve to eighteen hours earlier. The cervical abscess should have been drained through an incision anterior to the sternomastoid muscle; a light ether anesthesia would have sufficed; the boy would have been spared his agony of dyspnea; possibly the paralysis of the recurrent laryngeal nerve would have been averted.

SUMMARY.

Suffocative infections of the throat have been known and described since the days of early Greek physicians.

Until the nineteenth century an exact classification was impossible. Diphtheria, scarlatinal angina, quinsy, retropharyngeal abscess and other less severe diseases of the throat were grouped under the generic name *cynanche*.

Ludwig of Stuttgart described, in 1836, a particular form of phlegmon involving the tissues around the salivary glands and in the cervical region.

The anatomic planes of the floor of the mouth and of the neck, both muscular and facial, determine the spread of the disease.

The primary cause is frequently local affections of the mouth or trauma. The commonest finding is caries of molar or premolar teeth.

Streptococci are always present.

The pathology is that of a cellulitis, spreading from the tissues around the salivary glands to the opposite side of the throat and to the neck. Submaxillary and cervical tissues take on a boardlike hardness, but there is not a superficial edema.

Abscess formation takes place in over half of the cases.

The mediastinum is likely to be invaded.

Pulmonary complications are infarction, plastic and hemorrhagic pleurisy, bronchopneumonia, atelectasis.

The aggressive symptoms are dyspnea, alteration in the voice, dysphagia. The constitutional symptoms are those of toxemia.

Treatment is supporting and surgical. Intravenous therapy is not established as curative, but has been found of advantage in some cases. Surgical exploration, either for drainage of an abscess or for relief of tissue tension, should be early and searching. Pharyngeal abscesses are indicative of cervical sup-puration; their safest approach, when this is possible, is by an external route.

The mortality of reported cases is approximately 40 per cent.

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LVIII.

SPONTANEOUS HEMORRHAGE FROM THE TONSIL: CASE REPORT.*

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Those of us who are called upon somewhat frequently to remove the faucial tonsil have grown to expect the operation to be characterized by the loss of blood, slight or excessive, at the time of operation or afterwards, but to encounter hemorrhage when the tonsil has not been attacked, I feel, is to be resented almost as much by the attendant as by the patient. When facing such serious diseases as purpura, scurvy and the various disorders of the blood stream where the proportion of the corpuscles is seriously disturbed, spontaneous hemorrhage is to be expected, but not in an apparently healthy young person, such as the one whose case is to be described. Certainly here I found it somewhat disconcerting.

In the course of a routine examination of candidates for entrance in the school of nursing at Garfield Hospital in this city, I encountered a case where I considered a tonsillectomy to be urgent, inasmuch as the prospective nurse was certain to be assigned before long to the scarlet fever pavilion, where exposure to throat infection was, of course, inevitable. Despite my strong recommendation, the operation was not done, and for the next two years this young woman had mild but frequent infection of the tonsils. Two weeks before the hemorrhage with which this report is concerned she had a classical follicular tonsillitis, with the usual constitutional symptoms. After ten days she returned to duty, but one day felt a warm, salty taste in her mouth and expectorated a mouthful of blood. Being a stoical, conscientious type, she continued on duty, but was compelled to expel from her mouth a few drams of blood

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every half hour or so. This finally attracted the attention of the chief nurse, who relieved her of duty and had her throat examined by the intern on duty. As he could see no source of bleeding he thought it not serious, but the nurse insisted that her neck was swollen on that side and that a sense of pressure was constant; thus she came into my hands as chief of the throat service. There was a definite swelling of the neck at the angle of the jaw, and palpation gave a "doughy" feeling to the fingers, whose pressure caused her to feel the necessity for expectoration of blood. Her right tonsil, otherwise normal, was seen to be protruding somewhat into the lumen of the throat, and a crypt near the inferior part showed a fine stream of dark blood slowly escaping from its wide mouth. I wiped this out, and the instant that I separated the lips a gush of dark blood occurred, to be followed by total cessation of any hemorrhage for several minutes, when a thin stream again appeared and the collection of the excess in the deep cervical region followed. This procedure was kept up for nearly an hour under my observation, when I decided to make some attempt to stop it. The patient's observation of her own condition was rather striking in that she said that at first there was no swelling of the neck, but a constant flow of blood into her mouth, later, as soon as the neck swelled, the flow of blood stopped, to be followed by a feeling of pressure in the ear. I expressed, by pressure on the cervical swelling, a moderate amount of dark fluid blood. There was never any fever. The coagulation time of the blood was normal, nor was there any syphilis or tuberculosis. After expressing all possible collection of blood from the neck through the crypt I swabbed this out with 25 per cent solution of silver nitrate, which had to be repeated three times at intervals of 15 minutes. I held the swab in the bottom of the crypt firmly for three minutes the last time, and this sufficed to control the flow.

A week later I removed both tonsils under novocain adrenalin infiltration without any untoward incidents; especially was the hemorrhage negligible. The right tonsil was preserved, and the deep crypt, from which the blood had flowed, was found to penetrate into a plexus of vessels on the posterior pillar. At this point of the pillar at operation had been seen a bluish red congestion and a minute break, probably an ulcer-

tion. Thus the cause of the hemorrhage was, in all likelihood, an extension of the chronic inflammatory process in the crypt to the plexus of vessels and an ultimate ulceration into this vascular mass.

The patient has been in normal health ever since, and her throat has healed smoothly and firmly, showing nothing abnormal at the site of the ulceration; nor has there been any hemorrhage since operation.

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